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## CHIGAN HOSPITAL

THE NEW UNIVERSITY HOSPITAL.

# TRANSACTIONS

OF THE

# CLINICAL SOCIETY

OF THE

## UNIVERSITY OF MICHIGAN

OCTOBER, 1917—JUNE, 1918

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# TRANSACTIONS

OF THE

# Clinical Society of the University of Michigan

## Stated Meetings, October and November, 1917

**The President, JAMES G. VAN ZWALUWENBURG, M.D., in the Chair**
**Reported by REUBEN PETERSON, M.D., Secretary**

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## REPORT OF A CASE OF CONDYLOMA LATA AND CONDYLOMA ACUMINATA IN A PATIENT WITH SYPHILIS AND GONORRHEA.

JOSEPH A. ELLIOTT, M.D.
(From the Department of Dermatology and Syphilology, University Hospital, Ann Arbor, Michigan.)

This patient is a young lady, domestic, 19 years of age and single. She is an American and hails from Wheeling, W. Va. She was referred to this Department by the Department of Genito-Urinary Surgery on account of a four plus positive Wassermann reaction, having been under treatment previously for condyloma acuminata and gonorrhea.

Her present trouble began about three months ago when a sore appeared on the genitalia. The lesion was painful and has persisted. About one month ago lesions appeared in her mouth and approximately six weeks ago a large number of hypertrophic lesions appeared on the vulva and about the anus. Her hair has been falling out for two months. She has noticed no deafness, no loss of weight, and has had no bone or joint pains, and no headaches. When she came under observation she had had no specific treatment.

Examination disclosed a well nourished woman of fair appearance. The scalp shows a moth eaten alopecia. The eyes are normal and the pupils react to light and in accommodation. The skin of the face and body is clean except for a general, patchy alopecia over the arms and abdomen. Both the anus and vulva are covered with many hypertrophic papules, covered with a yellowish discharge. Some of the lesions are cauliflower in shape.

Over the pillars of the fauces are numerous ulcerated lesions covered with a grayish pellicle. There is a generalized shotty adenitis. The reflexes are active. The long bones are negative.

This patient entered the Hospital with a diagnosis of condyloma acuminata, due largely to the fact that the history had been taken previous to the examination, and it was found that she had a gonorrheal vaginitis. In view of this fact, I wish to discuss the differential diagnosis between condyloma lata and condyloma acuminata. We notice here a large number of verrucous lesions. On one lip of the vagina there is a large plaque, and on the inner surface there are a few papules. The interesting feature of the case is the large group of verrucous lesions. We can best understand the differentiation between condyloma acuminata and condyloma lata by conceiving a diagrammatic picture of the two lesions in cross section. Clinically the lesions may appear very similar. Pathologically they are decidedly different. By condyloma lata we mean the moist papules of secondary syphilis. By the term condyloma acuminata we refer to the pointed lesions which are prone to occur with any vaginal discharge. In order to make the differentiation we have to do more than a superficial examination. In condyloma acuminata the epidermis is thickened and thrown into folds and there is an increase in the size of the papillae of the corium. In practically all syphilitic lesions of the skin

the epidermis remains normal, or nearly so, but there is a decided change in the corium consisting chiefly in an infiltration of plasma cells and endarteritis. This pathologic change in the corium constitutes the induration of syphilitic lesions. In condyloma acuminata there is no change whatever in the corium, but there is a marked change in the epidermis, particularly in the prickle cell layer. Therefore, instead of the normal wavy epithelium, the epithelium is thrown into great folds, as in this case. So from observation, one could not distinguish the lesions in this case from condyloma acuminata. However, if by palpating we can determine infiltration, we can say that the lesions are syphilitic.

It is rather uncommon to see lesions of the mixed type. On the other hand, it is quite common to see condyloma acuminata in patients with a chronic gonorrheal discharge. The diagnosis of a mixed lesion therefore lies in establishing the fact that there is induration, indicating an infiltrate into the corium in addition to verrucous lesions. Here we have infiltration. This case therefore is presented as an example of a mixed lesion presenting the essential characteristics of both condyloma lata and condyloma acuminata. The lesions on the labium are purely condylomata lata. Those around the anus and posterior to the fourchette are mixed lesions.

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## THE CLINICAL APPLICATION OF ELECTROCARDIOGRAPHY.

George Edmeston Fahr, M.D.

(From the Medical Clinic, University Hospital, Ann Arbor, Michigan.)

Not long since I was asked by a colleague of excellent clinical and scientific training to decide by means of an electrocardiogram whether a case in which there was an undoubted lesion at the aortic orifice was complicated by a double mitral. Upon informing him that we could only get indirect evidence by this means, which would possibly weigh strongly in favor of, or against the diagnosis of a double mitral, he exclaimed, "Why I thought you could make a diagnosis of any cardiac condition with that wonderful instrument." At a scientific meeting in New York City a very short time ago. I was informed by a physician, whose name ranks very high in our profession, that "that ten days wonder, the electrocardiogram, is beginning to play out." These two remarks represent very well the two types of appreciation which the electrocardiogram receives from the great majority of the uninitiated in the medical profession of both continents.

Both the unbounded expectations of one group and the unwarranted depreciation of the other are due to a lack of understanding of the somewhat difficult physical and complicated physiologic principles which serve as the basis for our interpretation of the curves and to a lack of acquaintance with the clinical information which the electrocardiogram can give us. The electrocardiogram has come to stay in the study of cardiac disorders and it has become an essential part of the equipment for the diagnosis, prognosis and therapy of heart disease in every modern clinic throughout the world even though every question in cardiac disease can not be answered by it. It will not be possible to go into the physical and physiologic foundations of electrocardiography this evening as I shall confine myself strictly to an attempt to acquaint you with some of the information which the electrocardiogram supplies us in cardiac disorders. But before we plunge into the subject of clinical electrocardiography let us recall to mind that the electrocardiogram is essentially a graph of the spread of the wave of electronegativity which passes over the heart, but as this negativity is closely bound up with the excitation process, the normal electrocardiogram is really a picture of the propagation of the excitation wave from its inception in the sinoauricular node over the auricles and down the auricular septum into the auriculoventricular node and from here by way of the main branch, the right and left ventricular branches, and the outlying arborizations of the His-Tawara conduction system to all parts of the ventricular musculature. But the electrocardiogram is just as well a curve of the spread of the contraction process, for the course of the excitation wave in the heart muscles determines the course of the contraction wave. In other words, the electrocardiogram is a very direct means of examining the heart muscle. Before we examine our pathologic electrocardiograms, let us look at a normal one and review its prominent peaks and their significance.

Here is the electrocardiogram from a healthy young man who became a member of a college crew. The instrument is so calibrated that each millimeter along the ordinates equals a potential difference of one-tenth of a millivolt and the speed of the time marker which produces the ordinates is such that each scale division along the abscissae equals exactly 1/25 of a second. The upper curve is the so-called lead I. It is obtained by connecting the right and left hand with the galvanometer. The second curve is lead II, obtained by leading off from right hand and left foot. The lower curve is lead III, left hand and left foot being attached to the galvanometer in this case. This small upright wave is the P wave. It represents the spread of the excitation wave from the sinoauricular node over the auricles in the normal heart. When the excitation process starts at some other point or travels abnormally over the auricles the size and shape of this wave changes, that is, it points downward or is diphasic. From beginning of the P wave to the beginning of this sharp group, the Q R S group, the excitation pulse is traveling down the auricular septum to the auriculoventricular node and from here through the right and left branches of the conducting system into both ventricles. We therefore have an accurate measure of the conduction time here in our curve. This small downward peak is the Q wave and corresponds to the passage of the excitation process into the region of the ventricles about the papillary muscles. This upward pike is the R wave and is produced by the excitation process in its spread over the base of the ventricles. This downward pike is the S wave, and represents the shooting of the action-negativity into the apical portions of the ventricles. This normal Q R S group changes its form and size when the conduction process is slowed up or blocked along one of the branches of the conducting system, when one of the branches is lengthened in hypertrophy of the ventricle, or when an extrasystole originates in one of the ventricles. The whole ventricle is in excitation and negative at the end of the Q R S group. Now the excitation begins to die out and as it does not diminish in all parts at the same moment certain parts become more negative than others and this last wave, the T wave, results. Normally this wave is upright because the negativity disappears last at the base of the heart. The P and T wave are profoundly altered by vagus stimulation. The pike of the R represents the beginning of ventricular systole in the ordinary sense, diastole begins at the end of the T wave. The first heart sound comes just after the R pike, the second at the end of the T wave.

Let us first examine the typical electrocardiograms of valve lesions for, although the form of the electrocardiogram has no direct relationship to lesions at valves, yet as valve lesions are always found accompanied by changes in the size, distribution and quality of the cardiac musculature, we generally find typical and interesting changes in the electrocardiograms. Very often the electrocardiogram will furnish strong indirect evidence in the diagnosis of complicated or obscure valvular lesions at times when the state of compensation is such that typical murmurs are no longer present or percussion and X-ray are impossible.

In the endocarditic and arteriosclerotic forms of mitral insufficiency and mitral stenosis we usually get characteristic curves so that the diagnosis of a mitral lesion can usually be made from them alone. In mitral disease we have a massing back of the blood into the left auricle, the pressure at the end of diastole is greater than under normal conditions, the stretching of the muscle fiber stimulates to more powerful contraction, we get a dilated and hypertrophied left auricle. We should then expect to find an enlarged P wave in our electrocardiogram, representing this dilatation and hypertrophy.

Here is the electrocardiogram of a case of

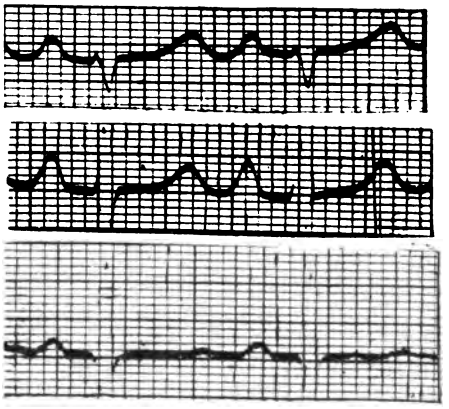

Fig. I. Mitral Stenosis. Large P. wave. QRS group downward in lead I, upward pointing in lead III, showing right ventricular preponderance.

well compensated mitral stenosis in a young girl of 18. (Fig. 1.) You see here this enormous P wave of 4 scale divisions or 4/10 of a millivolt. The normal P wave seldom reaches

two scale divisions. The P wave here is nearly as large as the R wave and larger than the T wave. This can only be interpreted as an expression of hypertrophy of the auricles. An enlarged P wave like this in mitral disease gives a relatively good prognosis for it shows the auricle is not fibrillating and that the cardiac impulse is formed at the normal site and is propagated over the auricles in the normal direction. An enlarged P wave can be used in the differential diagnosis of simple aortic valvular disease from aortic disease complicated with mitral failure. Large P waves are rarely found, Lewis says never, except where there is mitral disease with massing back of blood into the left auricle. The predominatingly downward trend of the Q R S group in lead I and the relatively high R in lead III shows a predominance of the right over the left ventricle, that is, a relatively greater enlargement of the right ventricle. This is general in mitral disease though as we know not necessary. In pure mitral insufficiency we nearly always have a large left ventricle. We frequently see a case of more or less pure mitral stenosis at autopsy with a large right and small left ventricle. In a double mitral, the relative extent of the stenosis and insufficiency as well as the power of compensation possessed by the left auricle are among the factors which determine whether we are to get relative right or left ventricular preponderance. I shall soon show you an electrocardiogram from a patient with a double mitral and relative left ventricular preponderance.

This lantern slide shows you another type of P wave often seen in mitral disease. The P wave is large and, in addition, of a complex form. It is long, diphasic and somewhat notched in lead I, diphasic and more distinctly notched in lead II, and of normal height but notched in lead III. This is to be looked upon as evidence not only of an hypertrophy of the auricles but also of sufficient change in the auricular musculature to produce a difference in the topography of the spread of the excitation and contraction wave in the two auricles. We have here I believe evidence that parts of the auricle are undergoing progressive change. This is verified by the electrocardiogram of this patient taken two and a half months later. The P wave is not as high as here, is longer and the notching has disappeared. Just a short time ago this patient came back to the Hospital suffering from extreme heart failure. The electrocardiogram and pulse showed the auricles to be fibrillating. I have another case under observation with this type of P wave, which changes its form from beat to beat, sometimes very markedly. I suspect that we shall get fibrillation of the auricles in this case soon.

Some years ago Samoilof of the University of Kiev published electrocardiograms from a case of mitral stenosis taken within intervals of about one year. In the first electrocardiogram, there is a large split P wave. The patient who came into the hospital decompensated, left well compensated after a short rest and digitalis cure. In the electrocardiogram taken about a year later upon the second entrance in the hospital he found the typical electrocardiogram of auricular fibrillation. The patient was now in such a condition that the process of restoring compensation was very difficult and the prognosis very dubious.

The electrocardiogram is of distinct value in the study of our cases of mitral disease. A large P wave excludes auricular fibrillation. In such a case there is ground for hope that by proper care auricular fibrillation, the greatly feared further alteration in the auricular myocardium in mitral disease, may be prevented or warded off for a long period. A study of electrocardiograms taken at intervals acquaints us with changes in the auricular myocardium.

A large majority of mitrals develop auricular fibrillation and the perpetually irregular and unequal pulse sooner or later. At the same time it must be remembered that auricular fibrillation develops under other conditions and it is of great importance to recognize it, as the prognosis in any given case is poorer, immediately fibrillation sets in and the therapy with sufficient doses of digitalis is indicated.

Here are the records from a case of mitral stenosis accompanied by auricular fibrillation. In this condition, which was first recognized through experimental electrocardiographic studies on animals and clinical electrocardiographic observations upon patients there is an incoordinate rapid twitching of small portions of the auricular musculature. These frenzied contractions do not aid materially in filling the ventricles. Besides, an irregular and rapid series of impulses are transmitted from the quivering auricles to the ventricles.

Here are the electric representatives of some of the twitches present in various parts of the auricles. The twitches are very irregular in their origin, extent and propagation and not all of them cause movements of the string of the galvanometer because of this abnormal ori-

gin and direction of propagation. Often absolutely no evidence of them can be found. Here they are very small. The frequency of the twitches is about 500 per second. Luckily not every twitch succeeds in getting an excitation wave passed down into the ventricles so that the ventricular rate does not get above 200 in general. In our case it is about 100, because the patient is still under the influence of a completed digitalis cure. A short time after the above was taken the patient was again put upon digitalis as he was beginning to suffer from dyspnea and dropsy. After a few days of digitalis a partial heart block was produced, the ventricular rate dropped to about 70 and the dyspnea and dropsy soon vanished. It is in producing heart block in auricular fibrillation and flutter that Lewis and MacKenzie find the most notable clinical effect of digitalis. Digitalis, if continued, produces complete heart block, the ventricle beating in its own rhythm. If the digitalis is continued premature beats or extrasystoles originate in the ventricles. The rate of the ventricles again increases. We get coupled beats in advanced digitalis poisoning.

Here is the electrocardiogram from a case in which the patient's heart had been fibrillating and was seriously decompensated. Digitalis was given in large doses. The heart rate was cut down at first from about 140 to about 70 after seven days' treatment. Then coupled beats appeared. Here is an extrasystole originating in the right ventricle. It is followed immediately by one originating in the left ventricle. Then there is a pause and the coupled extrasystoles begin anew. The rate of the heart is here about 120, so that the original digitalis effect of slowing the heart rate has been lost after excessive dosage. On the other hand the pulse rate was only 60 as there was only one pulse for each double extrasystole.

The first contraction originating in the right ventricle and spreading rapidly to the left gave a distinct pulse because the ventricle was full at this moment and the stretched muscle fibers could get up sufficient tension to overcome the pressure in the aorta. The second contraction beginning in the left ventricle and spreading to the right by conduction came at a time when the ventricles were nearly empty and therefore the fibers not stretched. The resulting contraction was not powerful enough to overcome the pressure in the aorta and no pulse resulted. It is worthy of note that a first and second heart sound was heard, followed very closely by a third heart sound at each coupled beat. This third heart sound was the first sound of the second ectopic beat. I show here a beautiful record kindly given to me by Prof. Einthoven of Leyden, Holland. This is a record of an electrocardiogram and the heart sounds recorded simultaneously. (Fig. II). Above we see first

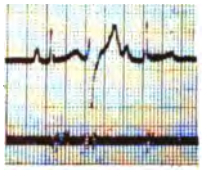

Fig. II. Extrasystole coming immediately after normal beat. Normal beat has normal first and second heart sound. Extrasystole has short first sound and no second sound. Heart sound curve is lower curve.

a normal beat accompanied by a first and second heart sound in their proper places; then there follows immediately after the end of this systole a new systole from an ectopic focus in the left ventricle. This heart beat has only the first heart sound accompanying it. Then after a pause of one-fifth second a new normal heart beat follows and this is accompanied by both first and second heart sound.

We occasionally find paroxysmal tachycardia associated with mitral disease in patients of an emotional type. In these cases there is a series of auricular extrasystoles originating in an ectopic auricular focus. These extrasystoles succeed one another at the rate of about 200

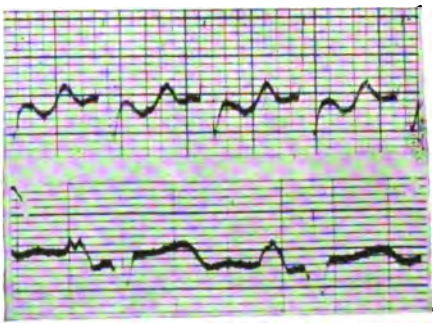

Fig. III. Paroxysmal Tachycardia. Upper curve taken during paroxysm, rate 180. Lower on normal day, rate 80. Note split P wave of mitral stenosis in lower curve, peculiar diphasic P wave in upper curve.

Here are the records from a case of mitral stenosis which I once had under observation for some months at Montefiore Home and Hospital in New York. The patient was very emotional and every attack of paroxysmal tachycardia was preceded by an emotional flare-up. The attacks came on suddenly, the pulse jumping with one bound from 80 to 200, and stop-

ping just as suddenly. During these tachycardias there were no other symptoms of decompensation than palpitation, precordial pain and inability to perform physical work. The patient at these times was very irritable and complained of queer sensations in her breast. Despite the fact that she had a mitral stenosis of about seven years standing with typical presystolic murmur, she never showed signs of congestion of the lungs during these attacks, though they sometimes lasted from twenty-four to forty-eight hours. Nor were there any signs of edemas or pulsating liver. Kidney function during these attacks was normal insofar as our functional tests can determine the same. Blood pressure was 120-130 systolic. I emphasize these facts here because they are in accordance with what we know of the dynamics of the heart as determined by Prof. Starling in his recent work. A normal heart, or in this case an abnormal heart with a good heart muscle, can keep up the normal blood pressure at a rate as high as this and throw out an adequate minute volume if the filling of the ventricles is adequate. Evans has shown that the heart fatigues more quickly at this high rate than at the low one since the metabolism is greater at the higher rate when the same amount of work is done by the heart upon the circulation. It is the fatigue which will ultimately change the elastic properties of this heart muscle and with this decrease in "tone" conditions will change for our patient. We shall then find all the classic signs of decompensation.

The lower record is lead II taken on a day when the patient was free from subjective as well as objective symptoms. The heart rate is 80. You see here the large split P wave of mitral stenosis denoting hypertrophied auricles, in this case, by the way, confirmed by fluoroscopy. The upper record is lead II taken on the following day when the patient was suffering from her attack of palpitation, precordial pain and uneasiness. The rate is here 200. Note that the P wave is now diphasic, and long drawn out, and partly coincides with the T wave of the preceding heart cycle. This diphasic P wave is the result of the ectopic origination of the impulse. An analysis of the other two leads compels us to conclude that the ectopic focus is in the left auricle. The cause of paroxysmal tachycardia in this case is to be sought in the auricular extrasystoles of very rapid rate originating in some abnormal point, undoubtedly in the left auricle. At the present state of our knowledge it would not be profitable to speculate on the ultimate pathologic substrata for these extrasystoles. They are always intimately connected with emotional states. We know that proper experimental stimulation of autonomic nerves supplying the heart may produce ventricular extrasystoles. Why should not the same thing cause auricular extrasystoles? I can only throw out this suggestion. At present nobody knows how these auricular extrasystoles are determined.

In this case, vagus pressure would not stop the tachycardia, nor would it cause an auricular ventricular heart block with a slower rhythm of the ventricles.

The electrocardiograms found in aortic disease have nothing very characteristic about them. The curves usually show left ventricular predominance, electrocardiograms of which I shall show later. Sometimes aortic disease is associated with auricular fibrillation or with extrasystoles, both of which are clearly shown by the electrocardiograms.

The various forms of arrhythmia, extrasystoles, tachycardia, auricular fibrillation and auricular flutter are the peculiar field of electrocardiography, for abnormalities in the origin and propagation of the auricular and ventricular contraction waves are clearly shown in the electrocardiogram when other graphic methods are silent or the records impossible of analysis.

The condition designated auricular flutter by Jolly and Ritchie is characterized by short or long periods of auricular paroxysm. It comes and goes suddenly and may remain for years. The auricular rate is from 200 up to 300 per minute. The ventricle may follow at this rate but there is generally a 2:1 or 3:1 heart block which causes a slower ventricular rate. The auricular beats originate in an ectopic focus. This is shown by the form of the P wave and by the electrocardiograms taken during vagus pressure. Vagus pressure usually succeeds in blocking the auricles when the beat originates in the sinoauricular node. In flutter the ventricle alone is blocked on vagus pressure, the auricle remaining in its old rhythm. I am now going to show electrocardiograms of auricular flutter under vagus pressure. (Fig. IV.).

The patient was on the TB service and the intern noticed a sudden increase in rate which startled him and which ceased all at once. The patient was sent down for an electrocardiogram. When she arrived the heart was beating at the rate of 115 per minute. The upper curve shows the electrocardiogram taken when the patient arrived at the heart station. The auricles are

beating at the rate of about 345 per minute, whereas the ventricle is beating at the rate of only about 115 per minute. As you will see, every auricular wave is not followed by a ventricular group. There is in this case a 3:1 heart block. Perhaps you will have some difficulty in finding the first auricular wave of each group of three because it is fused with the downward limb of the R wave. The R wave in this case should dip farther below the zero position of the string shadow and the first part of the T wave in this electrocardiogram is not a crest but a trough. The P wave of the auricle is superinposed upon this, elevating the trough above its normal position. If you will note that the tops of the succeeding P waves are four scale divisions apart, you will be able to locate the third P wave without any trouble.

Fig. IV. Auricular Flutter. Upper curve before vagus pressure, rate 115 with 3:1 heart block. Lower curve after vagus pressure, rate 85 with 4:1 heart block. Record taken by Dr. David Felberbaum, New York City.

The lower curve shows a 4:1 heart block, the ventricle beating now at the rate of 85. This was produced by pressing upon the vagus nerve in the neck. Vagus pressure did not affect the rate of the auricles because the auricular beats were not starting in the sinoauricular node where the vagus controls the rate of the normal auricle. The vagus stimulation on the other hand did succeed in blocking the passage of the excitation wave down the bundle three times out of four. Vagus pressure was now taken off and the ventricle began to beat at the rate of about 155, that is, the rate doubled because now only one impulse from the auricles failed to pass down into the ventricles. The shape of the P wave also indicates that the auricular contractions are beginning in an ectopic focus.

It is the increase in ventricular rate which we fear in our cases with degenerate heart muscle. With a good heart muscle, no venous engorgement, enlarged liver or edema will develop, a thing which always happens in those cases where the heart muscle has no reserve. In the case with good cardiac muscle, palpitation is the most noticeable feature.

By means of digitalis we can produce a ventricular heart block in auricular flutter so that the ventricle will beat at one-third to one-fourth the rate of the auricle. Large doses, continued long enough, will sometimes cause fibrillation. If the digitalis is now stopped in these fibrillating cases we sometimes see a return of the normal auricular beat, that is, the heart beat originates again in the sinoauricular node with a normal rate.

Auricular extrasystoles or premature beats are the source of arryhythmias in many cases. The auricular premature beats arise in abnormal foci in the auricles and give an abnormal P wave. Auricular extrasystoles are relatively harmless unless the rate of formation becomes extremely rapid, in which case tachycardia results.

We see here a case of auricular extrasystoles in a young boy of 14 years with a history of scarlet fever, diphtheria and rickets. (Fig. V).

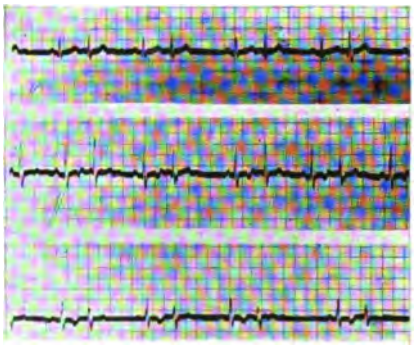

Fig. V. Coupled beats with pulsus alternans due to auricular extrasystoles. Note peculiar diphasic P wave in second beat of each group of two. Record of Dr. H. B. Williams, New York City.

He was conscious of a peculiar throbbing of the heart when lying down. Otherwise he is normal and able to attend to his school duties and play. We shall examine lead II first. We notice coupled beats in which the first shows a normal P wave and the second which follows immediately is of abnormal form. Please note the large negative P wave, showing that the impulse originates at some abnormal point in one of the auricles. From the pulse which has an alternans character, we might conclude that there was reason for a dubious prognosis. But a case like this gives a very good prognosis so long as the extrasystoles are as infrequent as they are here. Emotional states tend to increase the number of auricular extrasystoles and we are bound to advise against all excitement for patients showing auricular extrasystoles.

Here is another case with auricular extrasystoles even more infrequent than in the pre-

vious case. (Note that there is no compensatory pause). This patient complained of occasional very short attacks of palpitation on excitement. Otherwise he is normal. Undoubtedly the number of the auricular extrasystoles is increased during these attacks of palpitation. We have here the transition to paroxysmal tachycardia. We would speak clinically of paroxysmal tachycardia in our patient, if the attacks of palpitation were increased so that they lasted an hour or so.

We have here the record of an interesting case of paroxysmal tachycardia. The patient, born of humble parents, received a good education, being first in her class and very ambitious to become a teacher. Circumstances forced her to leave high school in her last year and take up a clerical position in the office of a cheap clothing manufacturer. The young lady had developed a taste for certain of the refinements in conduct. She liked to be with nice people of intellectual tastes, etc. The boss did not satisfy her requirements in this direction, his one ambition being to make money. The young lady became nervous, could not sleep nights and complained of a fluttering sensation in the chest. She visited several physicians but nothing abnormal in the heart rhythm was found because she was examined in the intervals between attacks. Finally the attacks became more frequent and a specialist examined her during a true attack of paroxysmal tachycardia and advised an electrocardiogram. This showed a paroxysmal tachycardia due to the formation of rapid beats in a new auricular focus. We see here four normal beats at the rate of 60 per minute, the impulse being formed at the normal sinoauricular pacemaker; then a series of rapid beats originating in abnormal auricular foci, finally a tachycardia of about 220 beats per minute. A case like this does not give a bad prognosis if the cause of the emotional states can be removed. The patient should be brought into a pleasant environment. We very often find cases of paroxysmal tachycardia associated with emotional states such as disappointment in love, failure of cherished plans, death of friends, etc. They generally stop when the cause of the emotion is removed or the emotion dies out. The danger lies in the paroxysms becoming permanent. If then there is a marked dilatation with edema, engorged liver, etc. the prognosis is bad. On the other hand many patients live for twenty years or more with their attacks of tachycardia. They feel well as soon as the attacks stop.

Not all paroxysmal tachycardias are of auricular origin, though it is true most are. (Fig. VI).

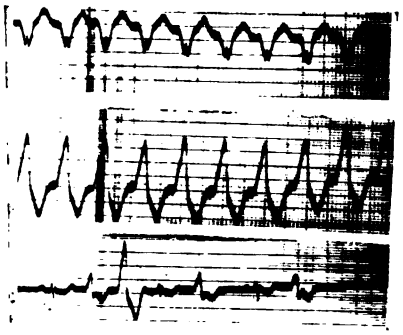

**Fig. VI. Paroxysmal tachycardia of ventricular origin.** Record of Dr. B. S. Oppenheimer, New York City.

We have here an electrocardiogram of a patient who suffered five days with this tachycardia of about 150 beats per minute. She was decompensated, had enlarged liver and congested lungs. She had been on digitalis outside the hospital but to no avail. Two injections of fifteen milligrams of morphine served to stop the attack, probably by stimulation of the vagus. Later on she had a second attack outside the hospital and this was stopped by morphine as before. This electrocardiogram taken during an attack shows in leads I and II a continuous series of extrasystoles of left ventricular origin. In lead III we have a series of coupled beats with one extrasystole of left ventricular focus. The second beat of the couple is also an extrasystole. In the intervals the patient has always shown isolated extrasystoles arising in the left ventricle. The patient is luetic and the daughter also. Antiluetic treatment has not changed her abnormal heart mechanism because the anatomic changes are already established. She also has an aneurysm of the ascending aorta.

We shall now look at some electrocardiograms of patients with extrasystoles of ventricular origin. This first lantern slide is from a patient with arteriosclerosis. Autopsy showed closure of some of the branches of the coronary artery. The patient suffered from angina pectoris. The extrasystoles originate in the right ventricle nearer the apex than the base. By means of the scheme of the equilateral triangle of Einthoven we can localize the focus of origination fairly accurately. I haven't the time to go into the method of localization here tonight. You will note that the extrasystoles in lead I and II are not followed by compensatory pauses, because the next following beat does not fall in the refractory period of the heart muscle. On the other hand the extrasys-

tole in lead III has a compensatory pause because the next following normal beat originating in the auricle would come during the time that this ventricle is still contracting and refractory. This patient showed extrasystoles every third beat when ocular pressure was exerted, showing that her extrasystoles are due to stimuli coming down the vegetative nervous system and are not due to changes in the heart muscle itself. Einthoven has shown this same thing in experimental morphine poisoning. The morphine poisoned dogs show numerous extrasystoles of right and left ventricular origin which cease on section of the vagus.

The next case is different. Only lead III is shown but it is sufficient for a diagnosis of left ventricular extrasystoles. The patient suffers from arteriosclerosis and has symptoms of angina pectoris. At autopsy, closure of some of the branches of the coronary artery was found.

Heart block gives characteristic electrocardiograms. Here is a 2:1 heart block in a patient with a positive Wassermann who improved upon mixed treatment. He suffered from cardiac asthma and with the improvement of the heart block the cardiac asthma also disappeared. The peculiar notching of the QRS group also indicates hindrance to the passage of the impulse down some branch of the conducting system. That it is only a partial heart block is shown by the fact that the ventricular complex follows the preceding auricular P wave after a definite pause which does not vary from beat to beat.

Here is a case of complete heart block which has persisted for eleven years. There is no history of syphilis and the Wassermann is negative. The patient had a slow pulse and fainting spells at 17. He now has fifteen a day. Nothing helps, neither digitalis nor atropine; he has thrombosis of the right femoral vein now. The ventricle beats at a rate of between 20 and 30. The intervals between the preceding P wave and the next QRS group are variable showing that the impulse to ventricular contraction is not conducted by the conducting system from the auricle. (Fig. VII).

Fig. VII. Complete heart block.

In the next electrocardiogram we see complete heart block. This is shown in lead I by the fact that the interval between the preceding P wave and the beginning of the ventricular complex is of variable length. The patient has had his heart block for many years and feels well in general. He gets attacks of Stokes-Adams syndrome. He has a large left ventricle, a systolic murmur. He had scarlet fever. The Wassermann is negative. The peculiar QRS group is worthy of notice. Normally lead I would be upright and lead III downward, pointing to enlarged left ventricle if the impulse came down along the normal path. Here an analysis of the QRS group by means of the method of the equilateral triangle shows that the impulse to contraction undoubtedly started in the left ventricular portion of the conducting system. Thus the left ventricle gets its negativity a little sooner than the right. This is further evidence that the block is complete for in enlarged left ventricle the right heart is negative first if the impulse comes down along the normal path.

Lately Dr. B. S. Oppenheimer of New York has called attention to a peculiar electrocardiogram associated with sclerosis of the subendocardial layers of heart muscle, that is, those fibers which contain much glycogen and which belong to the specific conducting system; especially the end arborizations of the same. (Fig. VIII).

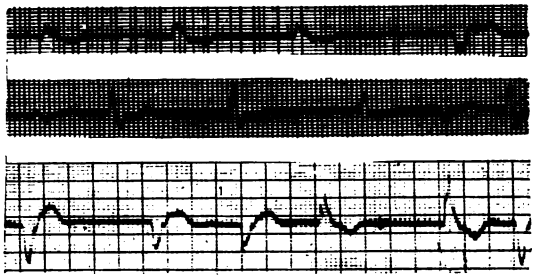

Fig. VIII. Split, prolonged and variable QRS group associated with degeneration of subendocardial layers of conducting system.

Here is the electrocardiogram of one of these cases. The auricles were fibrillating and no P wave is to be seen and the beat is irregular. Note the low, prolonged and split QRS group for this is the chief characteristic of this type of electrocardiogram. In the ordinary electrocardiogram only about 0.08 seconds elapse from the beginning of the Q to the end of the S. In this electrocardiogram we see as much as 0.16 seconds elapse before the completion of the electric variations which cause the QRS group. Note also that there are as many as six distinct pikes or notches to the QRS group here. This all shows that there has been some interference with the spread of the excitation wave over the entire inner surface of both ventricles. You will remember that I told you that the QRS represents the spread of the ex-

citation process from the time it leaves the right and left main branches of the His-Tawara conducting system to be conducted further by the subendocardial layer of endarborizations. Autopsy has shown that these conclusions are justified. In these cases we find sclerotic patches of degeneration occupying the place of the subendocardial muscle fibers.

We not only have interference with the conduction of the impulse to contraction in the endarborizations of the specific conducting system but also in the main branch and in the right and left branch. The former leads to heart block, a few electrocardiograms of which we have seen. The latter leads to a peculiar type of electrocardiogram an example of which I show here. Note the great height of the QRS group, its increased duration and the fact that the T wave in each lead has the opposite direction from the highest point of the QRS group. These are the characteristics of the electrocardiograms of bundle branch block and serve to distinguish it from left and right ventricular predominance which we shall discuss next. In bundle branch block the impulse comes down into one ventricle first, spreads over its inner surface very rapidly through the endarborizations and then more slowly through the rest of the muscle from within outward and across through to the other ventricle. When the negativity starts to die out, naturally it now does so earlier in that ventricle where the impulse first arrived and the result is this diphasic type of electrocardiogram. Bundle branch lesions are usually of luetic origin. Sometimes gummata press on the branch. At other times there is apparently no lesion demonstrable.

Predominance of right or left sided dilatation gives very characteristic curves. Usually it is said that right or left ventricular hypertrophy can be shown by the electrocardiogram. For various reasons which will come out in my discussion I prefer to speak of a predominance of the dilatation of the right or left side of the heart. Dilatation always precedes hypertrophy. When extra resistance is given a ventricle to overcome, it always begins by dilating, as we know from Starlings beautiful work on the factors governing the dynamics of the heart. This dilatation may soon be followed by hypertrophy. The increase in size of a cavity increases the length of the conducting system and its outlying branches in that ventricle and consequently the other ventricle receives its impulse to contraction earlier and becomes negative sooner. Therefore the direction of the potential difference in the heart is not the same when one side is negative first and the other follows later as when both are negative at the same time. It is the direction of the potential difference in the heart which determines the direction in which the R is turned in leads I and III. If the left ventricle is larger than the right then the impulse gets to the right side first and we have the R pike upwards in lead I and downwards in lead III as shown in this electrocardiogram which was taken from a patient suffering from chronic interstitial nephritis with blood pressure 270 mm. Hg. I shall now show the electrocardiogram of a patient with mitral stenosis whose right heart was known to be markedly dilated from X-ray findings and which on autopsy were confirmed. The right heart was much larger than the left and when I measured the length of the conduction path from a point where the left and right bundle separated to the papillary muscles in the right and left chamber and from here up to near the base of the corresponding ventricles I found the path in the right heart approximately 6 cms. longer. This is not the place to go into the subject of direction of the potential difference in the heart and the time relations of its turning. I shall say here that my measurements and analyses showed that the impulse got into the base of the right heart about 0.02 seconds sooner than into the left in the above case which would correspond to a conduction time about 2.5 meters per second. I have already published some of these analyses in *Deutsches Archiv für klinische Medizin* in 1915 and shall soon have a paper ready on the subject for a new publication. What I want to emphasize is that we can only determine a relative predominance of the right or left side of the heart with the electrocardiogram. For this purpose it is better than the X-ray in many cases. On the other hand we cannot say whether a heart is large or small if the relative enlargement on both sides is nearly equal.

It has been impossible for me to cover the whole field of clinical application of electrocardiography in the time at my disposal. I have only tried to show you some of the evidence which the electrocardiogram can give us. I believe, however, that I have shown the kind of evidence which the electrocardiogram can give us, so that you will all know what kind of evidence is to be expected from studies of the electrocardiogram.

Before closing I want to bring out one of the important uses of the electrocardiogram, a use

of which we will make more in the future, I am sure. In acute rheumatic fever the electrocardiogram often gives evidence of myocardial damage before polyarthritic and valvular symptoms develop. At a time when the only other lesion is in the tonsils we often find heart block or get extrasystoles. It takes a comparatively long time for valve lesions to develop. If we wait until they are fully developed valuable time will be lost for it often takes repeated infections to develop them. The good myocardium is of extreme importance in compensating the extra work put upon the heart by valve lesions. By prophylactic rest cures, graded exercises and baths we can often improve the myocardium and make it more equal to its future task. Or we may be stimulated to have tonsils removed or other foci of infection cleared up when we find that the electrocardiogram shows signs of damage to the myocardium. An electrocardiographic tracing will often bring home to us the serious import of otherwise seemingly negligible symptoms.

It can be said without exaggeration, that the electrocardiogram more than any other instrument has stimulated to an intense consideration of the very important subject of the mechanism of the normal and the diseased heart. The electrocardiogram is primarily a record of function in contradiction to the older method of auscultation and percussion which gives us primarily anatomic information. But I do not believe with Lewis that "graphic work has dealt a severe blow to the prestige of anatomic pathology." It is a worthy complement.

Allow me to show a slide of the laboratory of physiology in Leyden, Holland with its director Prof. William Einthoven the father of electrocardiography. To his systematic and untiring efforts the science of electrocardiography is due. Einthoven is the designer of the instrument by which electrocardiography is made possible. This instrument, the string galvanometer, may also be used for recording the heart sounds graphically, as well as the sounds of percussion. It has been used in wireless telegraphy and as a very sensitive recording seismograph. It could undoubtedly be used in the detection of submarines at a distance from the instrument. Prof. Einthoven is a pupil of the great Dutch ophthalmologist and physiologist, Donders. We see him here in his laboratory surrounded by his assistants, two of whom are in uniform as this was taken during the present war, and the Dutch are mobilized as well as all the other neutral peoples of Europe.

## LEUKEMIA CUTIS WITH DEMONSTRATION OF CASE.

JOSEPH A. ELLIOTT, M.D.

(From the Clinic of Dermatology and Syphilology, University Hospital, Ann Arbor, Michigan.)

The patient whom I wish to show tonight is an American Jewess, 48 years old, housewife. She comes to the Hospital because of an eruption over the body. Her family history is entirely negative, and her past history is negative. She has six healthy children.

Her present trouble began last winter when she states that worrying would bring on a backache in the lumbar region. She applied a liniment, and the backache left her in the spring. She has continued her housework up until six weeks ago, when she was forced to give it up on account of progressive general weakness and indisposition. Three months ago she noticed intense itching all over her body, followed by the appearance of a minute papular eruption. The lesions were pruritic, but not red. The papules have gradually increased peripherally, until now they are large plaques, itching only periodically. The lesions on the face appeared six weeks ago, and have been unaccompanied by subjective symptoms. The patient has been constipated for years, increased in the last six months. Four months ago she noticed a loss of appetite which has never returned. For the past three months she has had a cough which is very productive, yielding purulent sputum. She vomits about once a day, but believes this is induced by the coughing. There is no shortness of breath, but she complains of occasional palpitation of the heart. She has no fainting spells, and has noticed no numbness, but her feet become heavy after slight exercise. There are mild headaches. In the past three months she has lost twenty-two pounds in weight. She has had no night sweats. There have been no general pains, but of late she has noticed various prickling sensations over the cutaneous surfaces. Her ankles have been swollen once during the past six weeks. The patient gets up once a night.

Examination shows a fairly well nourished woman of average appearance. The scalp is clean. The pupils react fairly well to light, and accommodation. The skin of the face, especially on the left side, shows large blotches of a peculiar discoloration, having the appearance of a diffuse hemangioma. The skin of the trunk and extremities in general is of a sallow pale hue. Distributed over the body, and especially over the arms and trunks are numer-

ous infiltrated nodules. For the most part, these are rounded in outline, flat, discrete, raised and vary in size from that of a nickel to that of a silver dollar. In places the lesions seem to have involuted, leaving a brown pigmentation. The active lesions are not inflammatory, but of a slightly darker hue than the surrounding skin. In short, the lesions may be said to resemble very closely the pigmented wheals which one sees in urticaria pigmentosa. The mucous membranes are pale. There is a slight axillary adenopathy. The spleen is hard, firm, and palpable two finger breadths below the costal margin. The Wassermann reaction on the blood is negative.

Leukemia cutis may present itself in numerous ways. There is nothing particularly characteristic about the lesions. One of the conditions which has been described is spoken of as leukemid, and is not a true leukemia cutis, but is nothing more or less than a hemorrhage into the skin. This was first described by Friedrich, Ebstein and Fraenkel, and is the condition which appears on this woman's face. If a patient presents simply a diffuse hemorrhage into the skin we would naturally suspect that this was a septic process, and the only way to make a diagnosis would be from the blood findings. There is also a pruriginous eruption associated with leukemia which closely resembles prurigo Hebra. This is called prurigo lymphadenosis, and has been described by E. Wagner, Buschke and Briesendorf. It is, however, not characteristic of leukemia, but may occur in several conditions involving the lymph glands, such as lymphosarcoma, Hodgkin's disease, tuberculosis, and malaria. Further we may have a generalized erythema of the skin, and this also may be found in the other conditions which I have mentioned.

Lastly, we have two forms of leukemia cutis, the universal form, which is rare, and the circumscribed form, such as we have over the body in this case. This is the more common form, and is seen in acute leukemia, and particularly lymphatic leukemia. It is not pathognomonic because it is exceedingly rare, and resembles some other conditions, such as the early stages of mycosis fungoides. Here again the diagnosis is made from the blood findings. The location of leukemia cutis is fairly characteristic in most cases. The sites of predilection are the face, back of the hands, ears, and chin. Exceptionally we see the eruption over the entire body.

The pathogenesis of the condition has been a question of much dispute among dermatologists and pathologists. Some pathologists believe that it is a metastasis into the skin, a view founded on the fact that the cells are seen around the bloodvessels, where there is a proliferation. Others believe that it is a lymphoblastic proliferation of the tissue. Those who hold this view point to the fact that there are many mitotic figures.

The etiology of leukemia cutis is not known, but it is believed to be associated with some infection. Quite a number of observers have been able to isolate organisms and attempts have been made to demonstrate the tubercle bacillus, but without effect. Orth and Pappenheim have shown rods which were not acid fast, but which produced adenitis in guinea pigs. Arndt has found in some cases acid fast organisms, and he believes that many cases are due to the tubercle bacillus. It may be necessary to take out a pathologic section in order to arrive at the diagnosis. Of course in this case, the blood findings were fairly characteristic, and the biopsy shows a typical picture of leukemia cutis.

### DISCUSSION.

Dr. L. Harry Newburgh: I want to add to Dr. Elliott's paper the record of another case of leukemia of the skin. This case entered the Medical Clinic for the treatment of leukemia. The cutaneous manifestations were incidental. It was an example of so-called acute leukemia. The whole history ran for four months from the first symptom until death. The subject was a boy of 18, who first noticed increasing weakness and fatigue. Shortly after the initial symptoms, he noticed enlargement of both sides of his neck. These continued to grow, accompanied by more marked weakness and general debility. Next he began to suffer from dyspnea. The fourth event was swelling of the abdomen, chiefly on the left side. At this time he first went to a physician, who put him to bed and treated him for typhoid fever. There was no improvement and he came here two months after the beginning of his disease. Just before entering the Hospital he noticed that his face was discolored. His mother also spoke of the fact but there was very little made of it. He presented all the typical signs and symptoms of acute leukemia, running a continuous fever. There was marked glandular enlargement in the neck, axillae and groins; also a large spleen. He had a very large number of mononuclear cells in his blood and a moderately high white count. There was no question about the diagnosis. At entrance we noted that both cheeks, and an area extending to the chin and back as far as the ears were a dusky copper color. In addition to that the skin was thickened, differing in that respect from this woman. We were unable to feel any nodules. In addition to that he had a very marked, typical

purpuric eruption over the whole body. The skin of his face did not show the same color as the purpuric lesions. It was discolored and had a distinct thickening. The question came up as to whether this was a marked degree of hemorrhage into the skin, or a part of his general tendency to bleed. Dr. Wile at that time thought there was no question as to the nature of the skin involvement of the face. One of the interesting features of the condition was that it increased and decreased irregularly as time went on. When he first came in the condition became more marked. The copper hue and the thickening became more intense and then the condition decreased and later on increased again. He had nothing else in his skin which might be called leukemic. He had a great many hemorrhages from his mucous membranes and great crops of purpuric spots, but in no instance did he have anything which would at all suggest the conditions in his skin.

He also is an example of acute lymphatic leukemia. It is exceedingly interesting as a side issue that he was treated as typhoid fever because cases of acute leukemia are primarily treated for something else, such as myocarditis, sepsis, etc.

Dr. Quinter O. Gilbert: The differential blood count in the case presented tonight is as follows:

Reds 2,295,000, Whites 71,200, Hemoglobin 45 per cent. CI 45/45 = 1.

The count on 300 cells was as follows:

Polys 40 per cent., large lymphocytes 3 per cent., small lymphocytes 2 per cent., eosinophil polys 2 per cent., transitionals 3 per cent., large mononuclears 5 per cent., myelocytes 13 per cent., eosinophil myelocytes 2 per cent., myeloblasts 20 per cent., normoblasts 3 per cent. megaloblasts 7 per cent., total 100 per cent., degenerates 12 per cent.

This is a very peculiar differential blood count for a leukemia. The polymorphonuclear cells are decreased and the elements which are increased are the normoblasts, megaloblasts, myeloblasts and young myelocytes.

On the face of it, this is not then an ordinary leukemia in the sense that we speak of a lymphatic or myelogenous leukemia. I have seen only two bloods which approach this picture. One of them was in a patient whom we had here several years ago and whom I have held in reserve to write up because the blood was so peculiar and hard to differentiate. That was a case of very fulminating acute miliary tuberculosis. The count went up to a hundred thousand when the blood was loaded with gigantoblasts and cells from the bone marrow.

The first peculiar thing in the case shown tonight is the color index which is one. This is not like a leukemia because in leukemia there is a secondary anemia. It is more like hemolytic anemia and not true leukemia. Furthermore, in leukemia you never get so marked a disturbance in the myeloblasts, normoblasts and megaloblasts which make up 30 per cent. of the group. The other increases are all from the bone marrow. So this patient has some sort of a condition where there is a stimulation of the bone marrow and we know of such conditions in patients who have an acute infection or some toxic condition which stimulates the bone marrow. It is not a true leukemia in the nature of tumors. So it is a condition where there is a tremendous stimulation of the bone marrow producing a destruction in red cells giving almost the picture of a hemolytic anemia rather than a leukemia. It is very hard in such cases to differentiate between the undifferentiated myeloblasts, lymphoblasts and lymphocytes. I frankly admit that more differential stains should be used on this blood to differentiate the cells.

Dr. Mark Marshall: I saw this case with Dr. Elliott previously. I was of the opinion that I had a similar case two or three years ago, but after talking with Dr. Elliott, I learned that leukemia cutis was such a rare condition that I thought perhaps I was mistaken. In looking over the notes of the case and comparing the description of the lesions with the lesions which this patient presents, I feel quite sure I was mistaken. I think very likely my case was one of erythema nodosum.

I was interested in Dr. Gilbert's discussion of the differential blood count. I reported a case of miliary tuberculosis three years ago which had a differential count very much the same as this patient but the total count was not nearly so high. I think 30,000 was the highest count reached. The premyelocytic forms reached 35 per cent. That was a case of very acute miliary tuberculosis. It was diagnosed leukemia previous to the autopsy. The question as to whether we are dealing here with a leukemia seems to me should hinge upon the pathologic findings at autopsy. We don't know a great deal about leukemia at best. In leukemia one gets metastatic foci of abnormal cells such as these, while in an acute infectious process these metastatic collections do not occur, and they did not occur in the case which I reported. I saw the sections of the skin from this case which showed beautifully almost tumor-like formations within the skin itself.

Dr. Elliott: (Closing the discussion) In regard to the case which Dr. Newburgh reported, it seems to me that the case could very well be a leukemia cutis of the face and leukemia of the body, the latter consisting of diffuse hemorrhage into the skin. The differential count is particularly interesting. However, in practically all of the cases described there has been a marked increase of the lymphocytes and it has been distinctly lymphatic leukemia. Dr. Marshall's case may well have been leukemia cutis inasmuch as the lesions themselves are not pathognomonic. The lesions differ so that it is impossible to make a diagnosis from the character of the lesions. Arndt in his discussion of leukemia cutis classifies it under three different groups, aleukemic, with no increase of cells; subleukemic, with slight increase of cells; and leukemic, with decided increase of cells. All the lesions show the typical infiltrates into the skin.

# A REPORT OF A CASE OF HYDROPHOBIA.

HAROLD DEB. BARSS, M.D.
(From the Surgical Clinic, University Hospital, Ann Arbor, Michigan.)

The case which I shall report tonight is the second of two cases of rabies which have been cared for in the Surgical Clinic of this Hospital in the last fifteen months. I chose this case because of the extreme rarity of the disease in these days, and because the manifestations were so uniformly typical of the malady as recorded by all writers on the topic. It is interesting that of six men whose works I have lately read, three frankly stated that they had not seen a case themselves and were merely transmitting data derived from others. The other three neglected to state whether or not they had seen a case. It has been our good fortune (good from a scientific view only) to have witnessed two undoubted cases of hydrophobia. The first case was treated here on August 23, 1916, and I believe has been or will be carefully and comprehensively reported in the literature by Dr. Herbert W. Emerson who is in charge of the Pasteur Institute, and I shall not refer to it. The other is the subject of this paper.

It is not my intention to tire you with an exhaustive treatise on the subject. For this meeting I merely want to review briefly the outstanding features of the disease as recorded by most authors, and then compare these with the actual signs and symptoms as manifested in this case.

Rabies is an acute contagious disease produced by inoculation of a wound with a specific virus contained in the saliva of a rabid animal, usually transmitted by a bite. The exact nature of the infecting agent is not certainly known, but that it is some form of microorganism there can be no doubt. After inoculation, the virus is conveyed to the central nervous system by means of the peripheral nerves from the wounded part. Whether the toxins alone or the infective agent itself is thus transmitted is still unknown. After reaching the cord, the virus travels to the medulla, cerebellum and cerebrum; thence also along the spinal and cranial nerves and in this way reaches the salivary glands, especially the sublingual and submaxillary, and so renders the saliva highly infectious.

The period of incubation varies widely. The shortest period has been twelve days; the longest is not definitely known—several months to a year. The average is forty days. Incubation is shortest following bites of the face and wolf bites. The wounds are frequently insignificant, heal promptly and are forgotten.

Hydrophobia does not follow in every case bitten by a rabid animal. Roux, Babes and others estimate that only about 14 per cent. of persons bitten by a rabid animal develop the disease. The mortality has been considerably reduced since the development of the Pasteur treatment. The report of the Parisian Pasteur Institute shows that since its foundation there has been a mortality of 0.5 per cent. This treatment shows one twenty-fifth of the mortality which attends all other preventive methods.

In hydrophobia death is almost inevitable. Practically all cases in which it is alleged that recovery ensued were not true rabies but hysteria. An exception must be made in Murri's case. So far as I have learned Murri of Bologna cured the only case of true rabies which has recovered. This he did by injecting emulsions of cords of rabbits dead six, five, four and three days respectively.

In brief, the symptoms as recorded by most writers correspond almost exactly and are divided in three stages:

1. The prodromal period, during which the healed wound may itch or tingle, with sometimes neuralgic pains. Frequently there are no abnormal symptoms in the old scar. The patient complains of malaise, nausea, headache. Mental depression is the rule. There are sometimes chilly sensations, stiffness in the neck and slight respiratory difficulty due to incipient spasm of the diaphragm.

2. Period of excitement, characterized by increased anxiety; dysphagia because of pharyngeal spasms; intense hyperesthesia of the skin and all the special senses. Noises and drafts of air provoke sudden dyspnea and convulsions. The pupils are fixed and dilated. There are marked anginal attacks in these periods of apnea. Delirium is absent at first but may develop later. The seizures become more frequent. Salivation is marked. The patient is unable to swallow and so the thick tenaceous saliva drops away or the patient spits violently. Gradually the suffocative paroxysms which had been induced by external stimuli seem to come on spontaneously and the patient throws himself about and must be restrained.

3. The paralytic stage in which the patient sinks into a profound exhaustion. The pulse becomes rapid and weak, death resulting usually in coma with paralysis.

The entire course of the disease may be run in sixteen hours or may last four to five days, seldom longer.

The treatment must be prophylactic. Wide excision, or amputation of small parts, followed by the actual cautery, caustics, or germicides, preferably bichloride of mercury. If there has been a delay of some hours or days, the cautery is useless and may delude the patient into a sense of security. The treatment par excellence and the most rational is the Pasteur treatment, on which I will not touch in the present paper.

If the disease has developed, the only treatment now known is to relieve the seizures with morphine, hyoscine, chloral, or chloroform and give rectal water to alleviate the intense thirst. Curara has been tried because of its action in paralyzing the nerves supplying striped muscle, thereby controlling the convulsions. One cure through its use has been reported but it is doubtful if it was a true case of hydrophobia. It was tried in our previous case with little or no effect, save to control the convulsions. Antirabic serum is also given but save for the one known case has been of no avail.

Compare this textbook picture of the disease, with our case.

D. W. a school boy of 13, entered the Surgical Clinic of the University Hospital, October 1, 1917, at noon, with the following history: About the middle of last July he was bitten while trying to separate two dogs which were fighting. One dog was his pet, the other was strange. It was the strange dog which did the biting and so far as could be determined it was accidentally done. No one knows what became of the dog. The forefinger of the right hand was lacerated so that one stitch was taken and some powder dusted on. The wound healed promptly and nothing further was thought of it till September 29, when the boy felt an occasional momentary twitching pain in the bitten finger, slight nausea and a rather severe headache. The next day, September 30, the patient exhibited occasional attacks of dyspnea, some difficulty in swallowing, and a hypersensitiveness to cold drafts. On October 1 the difficulty in swallowing became more pronounced and the child was brought to the Hospital.

On entrance the patient appeared pale, eyes prominent and glaring. The facial muscles were very active producing frequent scowls, pouting of the lips, etc. The gait was somewhat stiff and spastic. When seated the boy sat huddled with his chin on the chest and the collar of his Mackinaw was pulled high around his head because, he said, the draft hurt him. The patient walked to his room and undressed himself. He said he felt well but couldn't swallow easily. Attempts to swallow seemed to interfere with breathing. He didn't see any need for going to bed but was willing. There were frequent exaggerated attempts at laughing and a sort of explosive method of talking, hurrying as though trying to get out an idea before one of the respiratory spasms should interrupt. He asked that the window be closed because the "air currents" worried him. His temperature was 102.6 deg., pulse 164, respiration 48. Antirabic serum was administered.

The patient remained about the same till 2 p. m. The only complaint was an increasing difficulty in swallowing. He could take only a teaspoonful of liquid at a time. The boy would take the spoon very carefully and slowly, would fill it and slowly approach his mouth and then suddenly throw the contents in, in an attempt to get it in and swallowed before the spasm. Each such attempt would excite an attack.

At 3 p. m. occurred the first general convulsion, which lasted about thirty seconds. The boy suddenly sat upright in bed and then threw himself back violently, clutched at his heart and begged the nurse to rub it hard. He called loudly for his mother, and from that moment he seemed to have an uncanny presentiment of impending death. From three o'clock on he kept calling for his mother, begging all the attendants to get her; shouting out at the top of his voice, declaring that if she didn't come soon, she wouldn't see her boy alive. "Tomorrow will be too late. If she doesn't come soon I'll be a stiff corpse when she does come." The convulsive seizures, tonic rather than clonic, succeeded at more frequent intervals. The neurologic examination at 3 p. m. by Dr. Camp showed the following: "The patient replies promptly to questions, seems excited. Occasional grimaces about the mouth accompany talking. No memory disturbance. Pupils dilated and fixed to light and accommodation. No palsies. heart rapid. Peripheral hyperesthesia."

Restraining sheets had to be applied at 3:20 p. m. The patient then became very restless, throwing himself around the bed. The convulsive attacks became more frequent and of longer duration. During the seizures in which the most prominent symptom was sudden spasm of the diaphragm with apnea, the patient cried out with pain over the heart and in terror begged the nurse to rub it, and called wildly for

his parents. His mentality was perfectly clear between attacks. He pleaded to have the restraining bands loosened. "I am such a little boy. Won't you get my mother? Don't you want to please me? Well, go to it then, get her, because if you don't, she'll never see her boy alive."

At 4:30 p. m. antirabic serum was again administered, followed by one-quarter grain of morphine sulphate hypodermatically. The spasms occurred still more frequently, the longest interval being seven minutes, the shortest, one minute. At the first hint of an approaching attack the boy would spring up to a sitting posture then throw himself back violently, the face contorted with frightful grimaces. The boy could no longer swallow and would spit forcibly. Any attempt to give liquids by mouth, or any sudden noise, would throw the lad into a spasm.

At 6:30 p. m. the second dose of morphine sulphate, grain one-quarter was given but was not effective. There was incontinence of urine. The bowels did not move. At 7:40 about forty grains of chloral was given per rectum; the exact amount could not be estimated as some of it was immediately expelled. The Murphy drip was started, but the water was not retained and that method was discontinued. The patient was still very excited and noisy till about nine o'clock when in an exhausted state the boy began to lie more quietly. A radial pulse could not be obtained, respiration was very irregular and shallow. There were only occasional convulsions during which the patient would strike out and claw at objects or persons near him. Now rational only at times. A reddish brown thick tenacious mucus dripped from his mouth and there were no longer attempts to spit.

At ten o'clock the patient became unconscious, the limbs rigid, the breathing stertorous, at times almost vocalized in a peculiar crowing sound. The rectal temperature at 11 p. m. was 107.8 deg. This comatose state continued till respirations ceased at 12:30.

It seems strange and most unfortunate that within the last fifteen months two patients both living within fifty miles of Ann Arbor should develop this most rare and terribly fatal disease. It seems hardly possible that the need of prophylactic measures in any suspected dog bite is still unknown by many persons. Had these cases occurred in the Upper Peninsula in some out of the way place where a doctor is not readily accessible we might excuse it. But when it occurs so close to this institution in a community where well informed men are practicing, something must be lacking.

The public is becoming educated to the dangers of tuberculosis and is becoming versed in the knowledge of the proper methods of prevention and cure of that disease. Tuberculosis clinics have been held frequently. Literature is spread broadcast and the press has aided remarkably in helping on this work.

It would seem that some more radical methods must be taken to get to the public mind the knowledge of the dangers of dog bites, and the measures necessary to avoid an occurrence of the disease. I believe that our doctors must be more alert to the possibilities of this infection, and the urgent measures that should be adopted to avoid it. The physician must keep ever fresh in mind the picture of the disease that he may more quickly diagnose the condition. I believe that the people should learn through wider notices in the press and through literature, the location in this city of a Pasteur Institute, what it is and how it may be used.

And more than this, there should be more stringent laws governing the keeping and care of dogs. In this regard, England has more stringent laws than any other country and rabies is an unknown disease there. Rare though it is here, it is all too frequent a visitor. Perhaps the dissemination of this knowledge is being conducted as widely as possible; but one who has seen the awfulness of the disease cannot help but hope that the time will soon come when there will be no more rabies.

For this State, I would make one other suggestion. The cost of such a course of treatment is almost prohibitive. It must be true that many persons have to take their chances, and run the risk of infection, because they cannot afford to take the treatment. There is in this city a Pasteur Institute where treatments are given for all suspected cases; but even at this Institute, the state requires that for all persons so treated, there shall be a deposit of twenty-five dollars ($25.00). One can readily see that for many persons even this price is absolutely prohibitive. The persons affected are not responsible for getting bitten, and they are not responsible for the laws which allow dogs to run free. It seems unjust that they should be required either to pay the twenty-five dollar tax, or else run the risk of infection. In my opinion, this treatment should be provided to citizens of the State, free from all tax, and

I earnestly place this suggestion for your consideration.

DISCUSSION.

DR. HERBERT W. EMERSON: There are only a few things which I wish to add, and they are to emphasize some of the lessons from these two cases. We have learned from experience to pay no attention to the history in cases of dog bites, as to whether the dog had rabies or not. Patients will come to you for treatment of bites and perhaps tell you that the dog was all right. We must not accept such statements without finding out the facts in the case. In this last case the patient reported to a physician and said that the dog was all right.

Secondly, in dog bites, one should resort to thorough cauterization with fuming nitric acid. Next to that, is the treatment of the wound with formaldehyde. The treatment of the patient after he develops symptoms of hydrophobia is chiefly palliative. There are three or four different treatments that have been recommended. I might call attention to the quinine treatment developed by Dr. Moon of Kansas City (*Journal of Infectious Diseases* XIII, 165) and used by Dr. Harris of St. Louis (*Journal A. M. A.* 1913, p. 1511) who reports a cure. This was probably a case of mistaken diagnosis. This treatment has been checked up in a number of laboratories and in our laboratory here by Dr. Cumming. It causes no delay in the development of the symptoms or in the death following in inoculated animals. It was checked up by Dr. F. S. Fielder of New York. One of his three cases yielded to the treatment but this was afterwards diagnosed as a case of hysteria.

Quinine and urea hydrochloride is given intravenously and intraspinally five to ten grains every few hours. Given intraspinally this is a dangerous method. In one case it was followed by an alarming collapse which lasted two hours. It had no apparent effect on the symptoms of the disease which proceeded three convulsions and paralysis to death.

The second treatment is the use of phenol in one or two per cent. solution given subcutaneously. This was first used about 1913 by Haberlin and one case of cure was reported which probably was a case of mistaken diagnosis because all cases since have shown no beneficial result of the treatment.

I have been more interested in the study of the treatment of this disease since I have seen these two cases, in the attempt to guide me in the treatment of the next patient should I be unfortunate enough to have one.

Hydrophobia is a protozoan infection, and inasmuch as a number of protozoan infections lend themselves to treatment, one might hope to find some substance which might offer some assurance of cure, but such has not been the experience of others.

DR. IRA D. LOREE: I would like to ask Dr. Emerson what disposition should be made of the animal and patient after the wound has been cauterized soon after its infliction with fuming nitric acid.

DR. HERBERT W. EMERSON: Have the animal locked up securely, for a dog suffering with rabies will get away where an ordinary dog will not. If the dog is all right at the end of ten days, don't worry. If the dog dies, the head should be sent to the Pasteur Institute and the patient should be sent to the Pasteur Institute. In any case in which you cannot get access to the dog, the dangers of the condition should be emphasized to the patient and if he wants to gamble with his life, he should be discouraged from doing so. In most cases, the patient or the parents of the patient have not realized that if the condition goes on until symptoms develop there is nothing that can be done.

DR. JAMES G. VANZWALUWENBURG: Personally, it seems to me rather futile to ask for more stringent laws because after all, a stringent law is bound to be a dead letter unless it is backed up by public opinion and public opinion is the feature which I think should be emphasized in this matter.

DR. BARSS: (Closing the discussion) I haven't much to add except in regard to the legal aspect. Rabies is practically unknown in England and Australia, due to legislation. Literature says that rabies is unfortunately all too common in America and we are lax in our legislation regarding dogs. I beg for a wider distribution of the knowledge regarding rabies. Often we doctors don't know what to do if we get a case of dog bite. We don't know how much security to take in immediate cauterization. We are coming to know about abortive treatment of tuberculosis and venereal diseases. We should know more about rabies. If you should talk with most of the doctors or lay people, they are absolutely helpless in knowing what to do in case of dog bite, or if they know that the dog was rabid, they don't know about Pasteur Institutes. If we have more widespread literature on the subject the laws are sure to come.

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## A CASE OF TYPHOID FEVER RECEIVING INTRAVENOUS INJECTION OF FOREIGN PROTEIN.

JOHN B. GRANT, M.D.

(From the Clinic of Internal Medicine, University Hospital, Ann Arbor, Michigan.)

I desire to report a crisis occurring in typhoid fever concurrent with the administration of foreign protein in the form of diphtheria antitoxin.

The patient, H. C., age 19, entered this Clinic the 20th of October, which was the fifth day of her illness, with the usual history of the onset of the disease. She presented a positive Widal test and blood culture. The course of the illness showed no complications except the marked tympanites of a severe case. The patient's temperature curve in the second week remained persistently above 104 deg. F. with a pulse between 100 and 110 and a white count averaging 3000. On the morning of the nineteenth day of the illness she complained of a sore throat. On examination this showed a membrane which gave a positive culture. At noon of the same day the temperature was 106.2 deg. F. and the leucocyte count was 5200. At 2 p.m. the temperature was 108 deg. At 2:15 p.m.

10,000 units of antitoxin was given intramuscularly, followed at 2:45 by 10,000 units intravenously. The sole manifestation of this was a chill beginning one-half hour after the intravenous injection and lasting forty-five minutes, accompanied by a slight delirium. The temperature began to fall immediately after the injection and had reached 99.1 deg. F. at 3 a. m. the next morning. Leucocyte counts made during this period showed a maximum of 6,000, two hours after the antitoxin was given. In ten hours it had fallen to 2,300, which has been constant since.

The intravenous injection of the antitoxin introduced a considerable amount of foreign protein into this patient's circulation. A number of observers have recently investigated the therapeutic value of the intravenous introduction of protein in typhoid fever. Various types of treatment have been followed out with specific and nonspecific protein. Also some investigators have used nonprotein substances.

Between 1913-1915 there were about 550 cases of intravenous injection of typhoid vaccine in the treatment of typhoid fever. McWilliams (1) draws the following conclusions in regard to these cases:

1. In fifty per cent. the disease was cut short one to several weeks.
2. A few deaths occurred where complications had already set in, especially hemorrhages.
3. No one type of specific vaccine is of especial importance.
4. Best dosage was 150 to 250 million.
5. The injection calls forth:
   First a leukopenia, later a leucocytosis with a slight increase in the eosinophiles.

Of the above results, the most important contributions were made by Ichikawa (2) and Kraus (3) who obtained termination by crisis in 20 per cent. of their cases.

Paulicek's (4) conclusions are of a special value because none of his sixty-eight cases came under observation until they were two weeks old and his mortality was 34 per cent., although the mortality in his control was even higher. He shows that to be of benefit the injections should be given in the first two weeks, for Lowry with Luksch and Wilhelm receiving their cases from the same trenches, only earlier in the course of the disease, had a mortality of only 11 per cent. Secondly, injections should not be given in complications of the circulatory system.

All these reactions, however, were due to the injection of specific typhoid organisms. Ichikawa and Kraus early determined that the reaction was not specific. The former used typhoid vaccine with equally good results in paratyphoid fever and Kraus obtained similar results in typhoid fever with colon vaccine. Lüdke (5) confirmed Kraus's results in eleven of a series of twenty-two cases by using colon vaccine. In ten others he used 4 per cent. deuter-albumose. In five of these he obtained a crisis followed by immediate convalescence. There were no fatalities. Lüdke found that as a rule in one-half to one hour after the injection there appeared a more or less heavy chill which lasted from one-half to three-quarters of an hour, followed by a rapid rise in temperature of one and one-half to three and one-half degrees F., which in forty-eight hours fell by crisis to normal. In some cases the temperature fell seven degrees F. in six hours. This period was accompanied by a drenching sweat; but in no case was there a detectable weakening in the pulse nor was there any untoward reaction. In contradistinction to the use of specific vaccine, there was never a leucocytosis. He concluded that the explanation was the same as a crisis of pneumonia.

Mittlander, (6) in his opinion, obtained improvement in some hundred cases by using intravenous injection of 1 cc. of 20 per cent. caffeine and 10 per cent. camphor followed by a saline solution, thus giving slight evidence that any agent not necessarily protein, which excited a chill, may bring about a sudden immunity to infection.

Janeway (7) in the Congress of American Surgeons and Physicians, refers to a termination by lysis of typhoid in a patient who had a chill following transfusion for hemorrhage.

Coming now to the explanation of the phenomenon of a crisis occurring after an intravenous injection of foreign protein in typhoid, we are flooded by a multiplicity of theories which leave us no better off than before. Gay and Claypole, (8) Gay and Chickering, (9) Jobling and Peterson, (10) Teague and McWilliams (11) are perhaps the ones who have attempted to investigate the mechanism most thoroughly. Teague and McWilliams have summarized the work of the others in a recent issue of the *Journal of Immunology*.

However, before giving their summary it would be of interest to touch briefly on some of the work they have been doing on the bacteriolytic power of normal human sera. Teague

and McWilliams found that one cc. of the sera of twenty-four non-typhoid patients would kill from one to one hundred million typhoid bacilli. In a series of forty-four typhoid patients, thirty showed the same bacteriolytic power. From this they conclude that typhoid fever cannot be a septicemia.

The same authors summarize three different views regarding the pathogenesis of typhoid fever:

1. It is a blood disease, a typhoid septicemia.
2. It is a primary infection of the intestinal follicles. The mesenteric lymph glands are next infected and finally the typhoid bacilli break through into the blood where they multiply, producing a secondary septicemia.
3. In typhoid fever the bacillus first finds its way from the alimentary tract to the lymphopoietic system including the spleen and from it, invades the blood stream. The presence of the bacilli in the blood represents merely an overflow from the lymph organs.

In view of their experiments, Teague and McWilliams rule out the first phase of a primary septicemia as unobtainable. Furthermore, since the serum of typhoid fever patients is only exceptionally of low bactericidal power, the view that the disease is a septicemia following the infection of the intestinal follicles and mesenteric glands must be discarded. *Ipso facto* there remains only a third hypothesis.

Having thus claimed to have arrived at a definite conception of the pathogenesis of typhoid fever the authors next proceed to outline the various existent theories of the mechanism of the cure of typhoid by the intravenous injection of protein. We shall not attempt to discuss them but leave them with you to consider:

1. Hyperleucocytosis which is chiefly upheld by Gay. (8)
2. Paralysis of the heat center suggested by Paltauf. (12).
3. A rapid mobilization of antibodies advanced by Bull. (13).
4. Setting free of nonspecific ferments, as suggested by Jobling and Peterson (10) in their theory that the cure is affected by the increase of the protease and lipase, the former producing a more rapid splitting of the toxic protein fragments to lower nontoxic forms, while the increase in the lipolytic ferments may have some influence in the destroying of the typhoid protein.
5. That fever is due to repeated anaphylactic reactions, due to the patient becoming sensitized to the proteins of the infecting microorganisms and as the latter enter the circulation, repeated low-grade anaphylactic attacks occur without producing complete antianaphylaxis. The sudden introduction of a large amount of antigen under these circumstances might completely desensitize the patient.
6. The theory of the authors themselves that typhoid fever is a local disease and that as the blood serum is strongly bacteriolytic, the intravenous injection of vaccine causes a more active passage of bacteriolytic substances from the blood capillaries into the "local lesions" with a destruction of the typhoid bacilli.

We present these views for your consideration without drawing any conclusions from them.

We would draw your attention to the fact that we are entering upon a new field in the therapeutics of infectious diseases.

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### DISCUSSION.

Dr. L. Harry Newburgh: There can be no doubt in your minds that we have approached an exceedingly difficult subject. There are, however, certain general conclusions which I think can be drawn. In the first place, we are at the beginning of what appears to be an exceedingly important method for the treatment of infection. Although the statistics are still confused, there seems to be no question

that the injection of protein into the circulation does in a general way affect favorably all sorts of infections. For instance, we know of the innumerable reports of improvement in various infections following the injection first of specific vaccines and then later of mixed vaccines. Those cases always did better when the vaccine was injected intravenously. There are reports of a large number of typhoid cases in which there was a very general decrease in the mortality and in the duration of the disease. About 30 per cent. of those cases had a crisis with rapid convalescence. I think we can also feel assured that this method is not a specific therapy. The earlier observers thought that they were working with vaccination. However, the injection of any protein into the circulation will produce the phenomenon which Dr. Grant has reported. In this latter instance globulin injected into a girl with typhoid fever causing a striking reaction, the temperature going from 108 deg. to normal in twelve hours.

There has been a great deal of discussion as to whether the improvement was produced by the leucocytes. In fact, the earlier workers had in mind the production of leucocytosis. Ludke has shown that one gets the same reaction in the absence of leucocytosis and we have here such a striking example. Of course, workers in this field have done what is human. They have obtained some results and have tried to explain them with a rational theory. Any hypothesis is neither good nor bad at the present. None of the explanations is complete. They are all confused, and for the present it is better to admit that we do not understand the mechanism. We should simply realize that this is an important new therapeutic procedure for all infections.

Dr. Paul DeKruif: It seems to me that my talking in a clinical society is rather out of place. The thing which impresses me most about the literature which Dr. Grant reports, is the total lack of experimental work on this subject. All kinds of opportunity present themselves to settle this question in a rational manner with experimental infections in animals, and that apparently has not been done at all. Besides the treatment of typhoid, remarkable results have been obtained in rheumatism by Miller of Chicago who used phylacogens, etc. Very recently Kraus from Argentina has used a normal beef serum in the treatment of anthrax with remarkable results. It seems to me that in all this work with nonspecific proteins there has been a lamentable lack of control work in the first place and in the next place, a lamentable lack of work with animals. I have no doubt at all that there was something more in this case than the effect of diphtheria. At the same time it seems to me that before this thing is applied in a wide-spread manner to therapeusis, there should be some decent experimentation upon it. Dr. Novy, whom I consulted about this question before I came, thinks that there might be one explanation made. I must confess that I have absolutely no explanation because the experimental work is so meagre that one cannot make one. Dr. Novy believes that there may be here a condition of immunity in these infections which is masked by certain blood colloids, and when you inject a substance to produce a chill, the substance may remove the masking substance and in that way liberate the immune bodies, which will control or abort the infection. And we had intended, if I had not been called away, to approach this subject in relapsing fever which is a particularly advantageous disease to use in animals because animals may get a third or fourth attack. At the same time you may show immune substances in the animals while the animals are suffering. We had intended to inject into such animals certain complex substances such as sugar, starch, kaolin, etc., and then see if there would be a decrease in the organism. For instance, joint infections in rabbits produced by streptococcus would offer a very good field for experimental approach to this question. All the six theories which Dr. Grant has mentioned should not be given the dignity of theories because there is so little to back them. I must say that of all the clinical work that has been done on the question, that of Gay is by far the most careful; but of course, he used specially treated typhoid organisms. It seems to me that you would be very fortunate if you were to get a nice typhoid epidemic of thirty or forty cases and go carefully about the thing and then be fortunate enough not to have a simultaneous occurrence of diphtheria. Then use straight serum globulin or normal serum and not diphtheria antitoxin, because in biologic questions it is desirable to use as little complicated materials as possible.

Dr. Quinter O. Gilbert: Dr. Grant said he didn't have a leucocytosis in this case. Technically he did have such. The patient had previously 2,000 white blood cells which went up to 6,000.

Dr. Harold deB. Barss: It might be interesting to state that last year we were treating a case of chronic actinomycosis of the abdominal wall in the Surgical Clinic in which the patient was running a temperature of 101 to 101.5 degrees. We injected Coley's mixed toxins intravenously and the patient developed a chill, temperature of 107 degrees, and became delirious. The next morning the patient's temperature was subnormal. His condition then improved remarkably and he left the Hospital, not entirely cured, however. The incident bears out the same finding of a foreign protein injected in a case of chronic infection.

Dr. Grant: I only wish to draw attention to the fact that in the literature where specific white counts have been made after injection of nonprotein vaccine, there has been no marked leucocytosis, as there has been after the injection of specific vaccines.

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## REVIEW OF A MONTH'S RADIOGRAMS.

James G. Van Zwaluwenburg, M.D.

(From the Clinic of Roentgenology, University Hospital, Ann Arbor, Michigan.)

The series exhibited comprise reproductions from 32 cases of general interest. In connection with a case of multiple dactylitis, the following observations may be made:

This is an example of a condition which we see comparatively rarely here but is quite common in some other clinics. It is one of multiple

dactylitis and the question is raised whether it is syphilitic or tuberculous.

I have seen so few examples of this condition that I am frank to confess that I am not well posted. I have already given the opinion that I thought it luetic. On reviewing the literature hastily, we incline to the opinion that it is tuberculous. As a matter of fact the literature is quite unsatisfactory, there being contradictions not only in the text but also in the examples reproduced. We gather that lues in its earlier stages is an almost purely periosteal process and the shaft of the long bones is always seen as a distinct sequestrum, while tuberculosis rapidly leads to a destruction of the sequestrum and the shaft disappears completely, as it is here shown. However, the same thing occurs in lues. I venture the suspicion that the determining factor in the production of this picture may be a secondary infection of the sequestrum by pus organisms, which is a common occurrence in either. Perhaps it would be safer to say that the differentiation can not be made with certainty.

In connection with a series of radiograms of the chest the following observations may be made:

I am showing this slide to introduce the subject of the significance of the bronchovascular tree.

The system of markings that we have called the "bronchovascular tree" is a striking feature of every good chest radiogram and has occupied the attention of every one that has done any amount of chest work. It has been the basis of a great amount of speculation and theorizing. A few years ago there was a very sharp controversy in America and in Europe as to the exact anatomic basis for these shadows, resulting in considerable experimental work on excised lungs. Perhaps the most extensive work was done in the Phipp's Institute by Dunham, Boardman, and Wolman. As a result of their observations the conclusion is reached that these shadows represent at least three components, viz., the bronchial tissues themselves, the peribronchial tissue including the lymphatics, and the bloodvessels and their contents.

Some of us now would like to go one step farther. Obviously it is conceivable and even likely that different pathologic processes react differently on these three components and it would be to our advantage if we could distinguish which was the most affected, whether the vascular tree, or the bronchial tree, or the lymphvascular system. Some evidence bearing on this problem has been accumulating for some time.

For instance, by means of the stereoscope it may be seen that in many cases these shadows have anastomoses in the periphery of the lung. Now, obviously, such shadows can not be bronchial in origin for the bronchi do not anastomose; they must be vascular, and probably venous at that, for the venous anastomoses are much more free and in the larger trunks than the arterial. Such a picture we have termed the reticulated type of B-V tree.

On the other hand, we sometimes see the tree dividing regularly into two branches, dichotomously. This occurs especially in long-standing cases of asthma where we know that pathologic changes in the bronchi are common and the inference is that these more nearly represent these anatomic structures. This type we have named the articulated type of B-V tree.

Sometimes we see a system of lines and markings that can not be classified in either of these types, they are irregular and disorganized; apparently replace the normal lung architecture, usually in an apex, and usually in the tuberculous. Dunham thinks these represent tuberculous infiltrations in the lymphatic system, at least in large part. Of course, when we consider the occlusion of lymphatics and subsequent diversion of the stream and its metastases, it is easy to understand its disorganization and irregularity.

Another factor that enters the problem is the blood in the capillaries of the lung. This represents a very considerable mass; as much as one-fifth or even one-third of the entire blood mass of the body may find lodgment in the lungs, it is alleged. This will naturally throw a deeper shadow over that portion of the chest where the lung is the thickest, namely, near the bases. Accordingly we find individuals that are relatively dense below, the density increasing progressively from above downwards. Ordinarily, the healthy chest is fairly evenly illuminated. This is particularly true of cases that have circulatory difficulties, and the roentgenologist knows to his regret that "heart cases" are notoriously "thick" and difficult subjects.

This first slide presents a case with a substernal goitre, with a large heart and an enormous dilatation of the bronchovascular tree. Unfortunately, it is virtually impossible to reproduce the finer markings of the lung texture on the lantern slide, and the anastomotic nature of the shadows can be demonstrated only by

means of the stereoscope. Judging from the size of the heart, we conclude that these are vascular in origin, but it is not at all clear that they are exclusively of that nature.

The next is an example of a frank cardiac case with enlarged bronchovascular tree below and a gradual increase in general density from above downwards. We interpret this as demonstrating a dilatation both of the large vessels and of the capillaries of the lung.

The next is a similar case. The plate is rather "thin" in quality because of an unexpected thickness that did not appear on the surface (capillary congestion), and the plate was undertimed.

Here is another cardiac case of a slightly different type. Not only is the B-V tree enlarged in the lower portion of the thorax but in the upper lobes as well.

We have additional information coming from cases not primarily cardiac, but inflammatory. For instance, last year I saw a nurse with a pain in the side. The only radiographic findings were an increase in the finer branches of the bronchovascular tree in the axillary region near the pleural border, probably about one inch beneath the parietal pleura. I reported at that time that presumably she had some sort of infection leading to a congestion at this point and that she might develop a pneumonia. Two days later she had developed a pleural effusion which I was able to follow radiographically until it disappeared. After her recovery I made another set of plates demonstrating that the appearance seen at the first observation had disappeared. I think, therefore, that I am justified in assuming that this represented a local hyperemia, and was an expression of a local inflammation.

Recently I have seen a somewhat similar case.

The next slide reproduces the radiogram of a little girl with acute attacks of dyspnea and coughing, whose side is fixed, the diaphragm moves little if any, the resonance is normal, all breath sounds are distant, no whispered voice, no fremitus, no expiratory murmur. Radiographically, we find this portion of the chest has about the same density as the other, but is almost free from shadow representing the B-V tree, as is easily seen by comparison with the opposite side. (We can not compare the lower portions of the thorax in this way because the cardiac movements serve to erase these markings in the left lower.) We are inclined to confirm the clinical diagnosis of bronchial obstruction. But that does not explain this asymmetry of the B-V tree. The other features are easily explained; the heart drawn over to the diseased side by the relative atelectasis. the fixation of the left diaphragm, without thickening of the pleural shadow or reduction of the width of the interspaces. I can see no other explanation than that there has been a change in the circulation of that lung, and the difference in the development of the B-V tree is a measure of the reduction of the blood content of the pulmonary vessels.

This last slide represents the conditions in chronic asthma. Here the B-V tree is distinctly of the articulated type.

These are shown here as examples of the differentiations we are trying to make in the study of our chest cases. I believe this is a more promising field than any other in radioraphy at the present time.

### DISCUSSION.

Dr. Joseph A. Elliott: (Discussing the dactylitis case) I was of the opinion that this was syphilitic rather than tuberculous. It seems to me that if this were tuberculosis, we would have involvement of the joints. A tuberculous process which would be inflammatory should show changes in the joints. It may be due to the fact that this child is so young that we get so much destruction of the bone. Textbooks say that tuberculosis destroys bones and syphilis does not, but due to the age of the patient, I am of opinion that we might well have such extensive bone destruction as there is here due to syphilis.

Dr. Cyrenus G. Darling: I have seen two or three cases similar to this one. I recall one case of a child which came to the Clinic ten years ago with an empyema. This was taken care of and drained for some time. and then afterwards developed a dactylitis I believe on the little finger of one hand, and I think that both hands were involved, one finger on each hand. This case was treated in the Medical Clinic and I think the diagnosis of tuberculosis was made. This child was under observation for four years and the two diseased fingers were very much shortened but never developed into an open sore. I cannot describe the condition of the bone because they were not X-rayed. I was under the impression that this was tuberculosis and I think it was so stated, the child being less than two years old at the time the condition developed. I think I have seen only two other cases in this Clinic besides the one I have mentioned, and those were both young children.

Dr. Van Zwaluwenburg: (closing the discussion) It seems to be distinctly "up to the radiographer" to justify himself in a great deal of this chest work and the problem is by no means simple. If you will read Osler you will find that he says, "more

than any others, radiographers need the salutary lessons of the dead house to correct their visionary interpretations of shadows, particularly of those radiating from the roots of the lungs." In all humility, however, I must differ with Osler. I see no certain way in which the dead house is going to solve our problems, because they are to a large extent problems in the actual circulation in the living lung, and when the individual dies, the circulation stops and the distribution of the blood is entirely changed.

I have tried to approach the question by means of Valsalva and the Mueller experiment with questionable results. I think of trying to solve the problem with animal experimentation. Perhaps I can persuade a surgeon to help me with a dog, and actually ligate certain vessels and see what happens. That would give us experimental evidence.

The time is past when the radiographer can look to his plates for empirical signs which shall tell him all that he cares to know, or can by *a prioi* reasoning reach a satisfactory explanation of the things he sees. We have reached the stage where we must analyze all of the evidence we find on all of our plates, determining the morbid anatomy and finally try to interpret them in terms of disease processes. If it were not for the excuse that our branch is young we should appear ridiculous, and that excuse will not serve much longer.

# TRANSACTIONS

OF THE

# Clinical Society of the University of Michigan

## Stated Meeting, December 5, 1917

**The President, JAMES G. VAN ZWALUWENBURG, M.D., in the Chair**
**Reported by REUBEN PETERSON, M.D., Secretary**

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## TIC DOULOUREUX AND ITS TREATMENT WITH A REVIEW OF THE CASES OPERATED UPON AT THE UNIVERSAL HOSPITAL IN 1917.

MAX M. PEET, M.D.

(From the Surgical Clinic, University Hospital, Ann Arbor, Michigan).

Neuralgia of the trigeminal or fifth nerve is much more common than neuralgia of any of the other nerves in the body. Three types are commonly considered. 1. Neuralgia minor, which often appears in the course of common diseases, such as colds, rheumatism and grippe, or following exposure, and is naturally more frequent in the fall, winter and spring. 2. Reflex or symptomatic neuralgia, often and probably correctly included under neuralgia minor, is found in cases of malaria, anemia, pregnancy, hysteria, sepsis, disorders of the stomach, acute intestinal infections, eye conditions, such as defective accommodation and astigmatism, accessory sinus diseases, and carious teeth. The last is probably the most common cause. One of the most frequent of the minor neuralgias commonly complained of is the sudden sharp pain on the side of the face and forehead which appears when drinking cold water or eating ice cream. The pains associated with the above neuralgias are usually relieved by hot applications, menthol, aspirin, and the relief of the disease giving rise to the irritation. 3. Neuralgia major or tic douloureux. This is the severe type which is practically intractible to ordinary treatment, is apparently not dependent on any previous coexisting disease, and is one of the most terrible afflictions which can come to a man, the pain being so fearful in its intensity that many have been led to suicide. It should always be distinguished from the ordinary forms, since the latter are amenable to simple measures and do not require the drastic procedures which alone can relieve the agony of tic douloureux.

The etiology of trigeminal neuralgia is obscure, and considerable divergence of opinion is expressed by leading writers. It usually appears in persons of good health, more frequently past the age of forty, and generally without any definite initial stimulus. Marked arteriosclerosis seems to have a causal relationship, although a few cases appear in young patients who have normal arteries. As a rule, no neurotic history is obtainable. In the majority of the cases seen personally, carious teeth, or even sound teeth have been considered responsible by the patients and nearly all have had the teeth removed from the side affected, but without permanent relief. Occasionally the pain ceased for a few weeks or months, but returned in greater severity and more frequent attacks. The sacrifice of sound teeth in cases of tic douloureux is entirely unjustifiable.

The results of the pathologic examination of excised gasserian ganglia and the peripheral branches have been disappointing. No lesion has proven constant, for, while very definite pathologic conditions have been found in cases of trifacial neuralgia, exactly similar findings have been shown in ganglia which have never caused trouble. The prevailing opinion at present seems to favor an ascending neuritis, of unknown etiology, beginning in the peripheral branches and finally involving the ganglion. This theory may explain the temporary relief some patients get after the extraction of a large number of teeth, the branch to this part being injured in some way, the return of pain being coexistent with the return of function in the nerve or the ascension to a higher

and uninjured level of the pathologic process. The fact that tic douloureux may be the only manifestation of a tumor of the gasserian ganglion should never be lost sight of.

Of the three divisions of the trigeminal nerve, the third or inferior maxillary branch is usually the first affected, the process often originating in the inferior dental, spreading slowly to the lingual and other branches of this division and perhaps not appearing in the second and third divisions for months or years after. In fact the first or ophthalmic branch is often spared. In one of our cases this division was the first involved.

The systemic effects of trigeminal neuralgia are marked. Many patients are seriously undernourished, and nearly all show some emaciation due to the inability to take food. They are frequently worn out mentally and in long standing cases are nervous wrecks from loss of sleep and the almost constant pain. Morphine habit is very common and in fact can almost be prognosticated, since these patients finally give out completely and being unable to bear the pain longer, seek amelioration in morphine as their only relief except suicide. One author points out that the mortality from suicide in these cases more than equals the deaths from the severest operative procedures for their relief. If for no other reason than the prevention of the morphine habit with its serious sequelae, patients suffering from tic douloureux should be referred to the surgeon early in the course of the disease, since in surgical operations alone can a cure be expected.

The pain of tic douloureux is usually paroxysmal in character, intermittent or periodic, and is confined to the distribution of the branches involved. The pain is intense, often of a burning nature, with lightning like onset; darts upward and backward along the course of the nerve, and may last scarcely a second. The intervals between paroxysms varies between the widest limits; frequently the sharp, shooting pain is followed in less than a minute by another, and then another, and this terrible agony may last for several hours, following which the patient may have relief for a few hours to many days. One of our patients had the pain quite regularly every three minutes. The paroxysms are often brought on by talking, laughing, eating, or the slightest irritation to the affected side of the face. Some patients are unable to shave on the sensitive side, as a paroxysm is certain to be induced. Exposure to cold or even a slight draught striking the face may bring on an attack. The first of our cases in this series would have a paroxysm when anyone entered the room where he was working. Spasm of the face on the affected side is not uncommon. One-half of the tongue may be involved, making eating and talking extremely painful.

Surgical treatment is alone to be considered in typical tic douloureux. The use of opiates should be condemned and other therapeutic measures are of no avail. The methods at our disposal are: 1. Alcoholic or osmic acid injections at the foramina of exit of the various branches on the face or in the course of the nerve, as within the mouth when the third branch is affected. 2. Neurectomy, preferably by the Thiersch method, either at the external foramina or along the course of the nerve. 3. Alcoholic injection at the cranial foramina of exit of the two lower divisions. 4. Neurectomy by exposure of the superior or inferior maxillary nerves at their exit through the foramina rotundum and ovale respectively as by Kocher's or Krönlein's methods. 5. Alcoholic injection of the gasserian ganglion through the foramen ovale. 6. Division of the two lower branches intracranially with interspersion of rubber tissue or a bone peg as in the Abbe operation. 7. Division of the ganglion and removal of the lower half together with the intracranial portion of the second and third branches as described by Hutchinson. 8. Removal of the gasserian ganglion by the Hartley-Krause, Lexer-Cushing, or Horsley operations. 9. Division or avulsion of the sensory root of the gasserian ganglion by the Spiller-Frazier or Kocher methods.

1. Alcoholic injections into the nerves at the mental and infraorbital foramina or along their course frequently give temporary relief; sometimes cessation of pain will last for four to six months. It is unusual to have it last longer. The disadvantages which this method possesses are: first, the relief when obtained is only temporary; second, subsequent injections do not as a rule give much relief, frequently none at all, or at most for only a very short time, thus necessitating the employment of some other procedure; third, the process may have extended beyond the area reached by the injection and no relief will be experienced.

2. Neurectomy is open to the same objections as peripheral alcoholic injections. The relief obtained is often of greater duration than after alcoholic injections, but rarely lasts more than a year. It is almost useless to repeat the operation when pain has once returned.

3. Alcoholic injections at the exit of the nerves from the cranium is quite satisfactory as a temporary measure. Permanent relief is scarcely ever obtained. The technic is not in itself difficult, but owing to the variation in skulls the nerve is not always to be reached and so a very short or no relief may follow the injection. Only the lower branches of the fifth can be reached by this method. The injection is usually quite painful.

4. Neurectomy of the superior and inferior maxillary nerves, the ophthalmic branch cannot be reached, at the foramina rotundum and ovale is difficult, rarely gives more than a year's relief, and in the case of the inferior division the motor root is practically always destroyed.

Alcoholic injection of the gasserian ganglion is strongly advocated by a few writers. It gives excellent results when the technic is carefully followed out and the alcohol actually enters the ganglion. Unfortunately it does not do this in many cases and simply makes an intracranial operation more difficult because of adhesions. Due to the great variation in the location of the foramen of ovale, it is often impossible to locate the opening and insert the needle. Many ingenious methods have been devised to locate the foramen, but none are infallable. Location by X-ray has recently been suggested. Even though the insertion of the needle through the foramen be simple, the difficulty of determining whether it has passed on through the ganglion or whether any other structures have been injured is so serious as to deter any but the most radical. In three of our cases the internal carotid lay exposed on the floor of the middle fossa, just posterior and external to the ganglion, without any bony covering. An injection of alcohol into the cerebral cortex, the middle meningeal artery or the internal carotid artery is not to be recommended.

6. The intracranial division of the superior and inferior maxillary branches according to the method of Abbe deserves attention, and when, owing to the condition of the patient, the least possible time should be consumed, the operation is indicated. The superior division cannot of course be reached with safety. In patients in whom the upper division is involved, the operation is therefore useless. In case it becomes subsequently involved either removal of the ganglion or section of the sensory root would have to be performed later. Unfortunately the relief expected is not always obtained, probably because of a pathologic condition in the gasserian itself. Occasionally also the rubber tissue becomes displaced or the nerves find their way around it and the pain returns, necessitating another operation. The motor root of the fifth is practically always destroyed.

7. Hutchinson's division of the glans and removal of the lower portion together with part of the lower two divisions has given excellent results, but at least in my hands is as difficult as section of the posterior root. The motor root is always destroyed and where the superior branch is not affected at time of operation, the possibility of later trouble is always present. With involvement of this branch the operation is useless. The arteries supplying the gasserian enter it from beneath and sometimes troublesome bleeding results during its partial or entire removal. The principal advantage of this operation, which it also shares with the Abbe, is the absence of anesthesia in the eye.

8. Total extirpation of the gasserian ganglion by any one of the approved methods guarantees absolute relief from pain. Its disadvantages are, troublesome bleeding, danger of injury to the cavernous sinus, third, fourth and sixth nerves, destruction of the motor nerve, and time consumed as compared to simple section of the sensory root.

9. Division of the sensory root between the gasserian ganglion and the pons offers the following advantages over all other methods of treatment: first, less hemorrhage as the troublesome bleeding from the vessels which enter the under side of the ganglion is eliminated; second, the third, fourth and sixth nerves and the cavernous sinus are much less liable to injury; third, it is possible in some cases to isolate and preserve the motor root, thereby preventing paralysis of the muscles of mastication; fourth, the cornea seems less liable to ulceration, possibly because of trophic nerves passing from the ganglion to the eye. Division of the sensory root gives permanent relief from pain in all branches of the trigeminal nerve.

In brief, the operation consists of the exposure of the skull above the posterior half of the zygoma by a "question mark" incision, with reflection of the musculocutaneous flap. The skull is trephined, the opening enlarged to about three centimeters in diameter, the dura elevated by blunt dissection, and the middle meningeal artery isolated and divided. The inferior maxillary branch then appears at its exit through the foramen of ovale. The dura is separated from the ganglion in a medial and posterior direction and the sensory root exposed

as it passes over the petrous portion of the temporal bone to the pons. With a blunt hook the sensory root is elevated and separated if possible from the motor nerve. It is then divided and the wound closed in layers.

The mortality from operations on the gasserian ganglion is now about 3.7 per cent., a risk favorably comparable in every way with nearly every other major operation. Recurrence when the ganglion is actually removed in toto or where the sensory root is divided, is practically unknown. Such a recurrence indicates central origin of the pain.

The paralysis of the muscles of mastication on one side which follows division of the motor root is not very distressing and the patient soon forgets the deformity. The anesthesia of the face is such a grateful relief from the hypersensitiveness and pain that it is never complained of. The ocular symptoms are the most aggravating, but even when the most serious conditions develop, necessitating the enucleation of the eye, the patients state that they are more than willing to pay that price for the relief obtained.

In this Clinic the division of the sensory root is preferred to all other operations, for the reasons given before. It has been our experience that the extracranial operations and alcoholic injections are only palliative and that an intracranial operation must ultimately be performed. It therefore seems advisable to perform the radical operation in preference to the others, and as early as possible, before the patient's general health has been destroyed and before a drug habit has been formed.

The following cases have been operated upon the past year in the Surgical Clinic of the University Hospital. We wish to thank Dr. Camp of the Neurologic Department and Dr. Lyons of the Dental Department for referring some of these cases to us; to the above men and Drs. Parker and Prangen of the Ophthalmologic Department and Drs. Barlow and Furstenberg of the Otologic Department for examination and advice in the care and treatment of these cases.

Case I. No. 5919. Mr. E. W., age 34. Mail clerk. Family and personal history negative. Present illness began five years ago with pains in right lower jaw, apparently located in the molar teeth. The pains came on two or three times a day and lasted one to three minutes. Two years later had alcoholic injection in the infraorbital and infradental branches of the fifth nerve. Pain was relieved for only a short time. Three months after the injections he was operated upon and the inferior maxillary branch avulsed at a point in the ramus of the jaw. No further pain was experienced for seven months when he had a recurrence of his former shooting pains in the right upper and lower jaws radiating particularly along the zygoma to the ear. Accompanying this he had a facial spasm on the right side. The pain has increased in severity, the attacks in frequency and for the two weeks preceding admission his condition has become almost unbearable. The attacks now appear from one to three minutes apart. An attempt to eat, speak, or make any motion with the mouth brings on a paroxysm of pain and a spasm of the right side of the face. As soon as the pain returned after avulsion of the nerve, alcoholic injections were again made but without relief. The pains have now persisted for three months. Physical examination shows a somewhat emaciated individual with marked spasm of the right side of the face, coincident with the attacks of pain. The hand is clapped suddenly to the face and the head bent to the right during the paroxysms. These appear when any attempt to answer questions is made. Neurologic examination negative except for partial facial paralysis, the result of the previous operation. X-ray negative. Wassermann negative.

March 5, 1917, operation by Dr. Peet under ether anesthesia. An intracranial operation was performed with section of the sensory root of the gasserian ganglion. No unusual difficulties were met. The motor root could not be distinguished and was cut with the sensory. Convalescence was rapid; complete anesthesia was present in the distribution of the right fifth nerve, and the patient was discharged cured nine days after operation.

Subsequent history. Patient has been entirely relieved of pain, but developed a slight keratitis which was treated successfully in the Ophthalmologic Department. He has gained rapidly in weight.

Case II. No. 6155. Mrs. M. C., age 52. Housewife. Family history negative. Personal history negative except for five miscarriages each at five months and a choreiform attack consisting of jerking movements of the right arm and hand which lasted for three months and terminated at birth of her first child. Present illness began about four years ago with pain on right side of face which appeared while talking or eating. Pain is sharp and lancinating, comes on in paroxysms and radiates all over right side of face. Had alcoholic injection by Dr. Camp in 1915 with some relief. Was operated upon in the Surgical Clinic two years ago, at which time resection of the inferior dental nerve was performed with temporary relief. The pain has constantly increased in severity and now interferes seriously with sleep. All teeth were removed without alleviating the symptoms. Alcoholic injection one week ago gave no relief. Patient says for the past four years she has practically been unable to talk. Physical examination negative. Neurologic examination negative except eyes react somewhat sluggishly to light and there is a slight lateral nystagmus in both eyes on looking to right and left. Wassermann negative.

April 7, 1917, operated upon by Dr. Peet under ether anesthesia. The sensory root of the gasserian ganglion was divided on the right side. Motor root was uninjured. Complete anesthesia on right side of face with loss of pain. Subsequent history. On the seventh day patient developed a

partial paralysis of the sixth nerve. This was still present when patient was discharged cured, April 27, 1917.

Case III. No. 6324. Mrs. M. B., housewife, age 41. Family history negative. Personal history. Patient has had hard headaches for years, but says the pain in the face is entirely distinct. Was operated upon at the Mayo Clinic seven years ago and a diagnosis of abdominal tuberculosis was then made. Apparently has recovered from this trouble. Appendix has been removed and the gall bladder drained. Present illness started suddenly three years ago with severe continuous pain on right side in region of lower molar teeth. Pain varied in intensity, being very severe at times, so that morphine was necessary, but the pain never ceased entirely. This lasted for a year when she had all the teeth, twenty-eight in number, removed at the same time, under chloroform narcosis. The pain was completely relieved for five months, when it returned in the same region and with even greater severity. In May, 1916, the inferior maxillary branch of the fifth nerve was avulsed through an incision at the angle of the jaw. This gave complete relief until December, 1916, when the pain gradually returned, appearing at intervals of one or two days. In March, 1917, the pain became practically continuous, one paroxysm following another in rapid succession. It was also much more severe and sleep could only be obtained by the use of large doses of morphine. She had an alcoholic injection last week but without relief. The pain now covers the entire cheek up to the level of the zygoma and down under the inferior maxilla to a point one inch from mid-line. Right side of the tongue is involved, but the nose, eye, and upper portion of the mouth are free. She still has the headache, most marked over the anterior vertex. Physical examination negative except for considerable emaciation. Blood pressure, systolic one hundred ten, diastolic seventy. Wassermann negative. Neurologic examination shows fine tremor of tongue which protrudes slightly to the right. Protrusion of tongue is very painful. Bilateral ankle clonus, no Babinsky. Patient is almost unable to eat or sleep.

April 7, 1917. Operated upon by Dr. Peet under ether anesthesia. Sensory and motor root of gasserian ganglion divided. The motor nerve could not be distinguished even after section of the root. Patient was immediately relieved of the excruciating pain and had complete anesthesia of the right side of face. Lagophthalmos and keratitis of the right eye developed immediately and were treated in the Ophthalmologic Department by Dr. Prangen. Patient discharged cured April 21, 1917, with keratitis much improved.

Subsequent history. Keratitis has returned at intervals. Patient is free from all pain and has gained fifty pounds in weight.

Case IV. No. 6679. Mr. I. F., age 52. Farmer. Family and personal history negative. Present illness; about two years ago he noticed a sharp "cutting" pain which came on at times when he caught cold or otherwise irritated the right side of his face. The pain starts at the median line of the lower lip and radiates backward following the mandible up to the auricle. It is quite intense over the malar bone. Soon after the attacks commenced all the teeth were extracted, but without relief. The pain was often brought on by talking, eating, or shaving the right side of face. It appears at irregular intervals, often, many times a day, and may waken him at night. The pain is often burning in character, especially a burning sensation in the upper lip. The paroxysms are of great severity and are described as sharp and shooting, as if a red hot knife was thrust upward through the face. No remedies give relief. Physical examination negative. Neurologic examination negative except for a hyperesthesia of the right side of the face beginning at the middle of the lower lip. This hyperesthesia has been worse than at present. The tongue protrudes to the left of the median line. X-ray examination shows the neural canal is normal and there is no evidence of pathology. The eye examination shows early arteriosclerotic changes and slight edema of the retina. Wassermann negative.

April 21, 1917. Operation by Dr. Peet under ether anesthesia. Inasmuch as the pain was confined to the lower two branches of the fifth nerve, only the lower two-thirds of the sensory root on the right side was divided, thus insuring some sensation in the eye. The motor root was isolated and was not divided. After operation complete anesthesia of the cheek, upper and lower lips existed. This anesthesia stopped sharply at the mid line of the lips. A herpes of the upper lip on the right side developed after the operation and lasted about five days. Eye examination by Dr. Parker on April 30th showed anesthesia of the outer half of the right eye. The patient was entirely free from pain and was discharged cured, April 30, 1917.

Subsequent history. On July 12, 1917, the patient returned to the Hospital complaining of a severe burning sensation on the right side of both upper and lower lips. The pain had not returned. He was examined by Dr. Camp who made the following report: "The examination shows a weakness of the right motor fifth, sensory disturbance in the distribution of the right fifth, including the first division which was formerly (i. e. immediately after operation) exempt. I would be of the opinion that following the operation the patient has developed an inflammatory change in what remains of the gasserian ganglion and that this is the cause of his present symptoms."

It seems peculiar that a burning sensation is felt on the right side of the lips and yet this area as well as the remainder of the face supplied by the superior and inferior maxillary divisions are absolutely anesthetic to pain, touch, or temperature. He was given aspirin and asked to return later for another examination.

August 19, 1917, patient returned to the Hospital complaining of a severe burning sensation on the right side of the upper and lower lips and adjacent side of the face. The anesthesia is complete as before. It was suggested that an inflammatory process had involved the central end of the sensory root or that it was caught in scar tissue. This explanation if it applied to cut ends of any of the branches distal to the ganglion would seem feasible Such symptoms referable to injury of a sensory root proximal to a ganglion are unknown to me. Since the patient insisted that something be done another

operation was performed August 20th and the ganglion removed together with the larger part of the central end of the sensory root. Following this, complete anesthesia of the entire distribution of the fifth nerve was present, the burning sensation was completely relieved and the patient was discharged cured fifteen days after operation. In October the patient returned with a rather severe conjunctivitis in the right eye and slight burning on the right near the middle of the upper lip.

A satisfactory explanation for the burning sensation in the lips after section of the sensory root, and again after removal of the ganglion, is difficult to make. Probably it is of central origin, but if so, its temporary relief after the first operation and complete relief after the second, except for the slight return in the upper lip needs elucidation. The shock to the central nervous system from the intracranial operations may explain this.

Case V. No. 7055. Mrs. J. F., age 68. Housewife. Family and personal history negative. Present illness began seven years ago with "jerking" pain over the right side of the upper lip which radiated backward to the auricle and downward to the margin of the jaw. Pain at first appeared at monthly intervals, then became more frequent until at present it is almost continuous. History of previous treatments is indefinite. Physical examination shows a very much emaciated, feeble, old lady, in terrible pain. No pathologic conditions evident by palpation or auscultation. All teeth have been removed. Neurologic examination negative. Wassermann negative.

June 2, 1917, operation by Dr. Peet under gas anesthesia, because of her very feeble condition. The sensory root of the gasserian was divided. The motor root could not be distinguished and was necessarily cut. Patient was discharged cured, with complete anesthesia of right side of face and no pain, six days after operation.

Subsequent history. The patient rubbed the cornea of right eye rather vigorously and developed a conjunctivitis which cleared rapidly and without leaving any ill effects.

Case VI. Mr. J. P., age 65. Watchman. Family history negative. Personal history negative except for hay fever for the past twelve years, which usually appeared the middle of August. Present illness started six years ago with sharp pain in the right cheek which lasted about thirty minutes. The attacks have returned frequently, coming on at shorter intervals until at present the pain is almost constant. He describes it as dull and aching in character with sharp shooting pain of great severity, coming on every half hour and lasting about three minutes. The attacks are growing more severe in character. Pain is confined to the lower two branches of the fifth, the ophthalmic never being affected, although the right eye is tightly closed during an attack. Neurologic and physical examination negative. Wassermann negative.

June 16, 1917, operation by Dr. Peet under ether anesthesia. Since the pain was confined to the supramaxillary and inframaxillary divisions, only the lower two-thirds of the sensory root was divided. The motor root was not injured. The patient was discharged cured, six days after operation. Complete anesthesia of the lower two branches with normal sensation in the conjunctiva and forehead was present. Pain was completely relieved.

Subsequent history. No complications have arisen. Pain has not returned.

Case VII. No. 7132. Mrs. E. H., age 46. Housewife. Family history negative. Personal history negative except for a "nervous" attack fourteen years ago which confined her to bed for two weeks Prsent illness began four years ago with pains on the left side of the nose and cheek. The attacks came on every spring and lasted all summer. Recently the lower jaw on the left has also been affcted. All teeth were removed ten years ago. She has had numerous alcoholic injections without relief but has not used morphine. Neurologic examination by Dr. Camp was negative. Wassermann negative. X-ray showed chronic sinusitis of left maxillary antrum and ethmoids. Examined by Dr. Canfield who did not recommend treatment for the otologic condition.

June 27, 1917, operated upon by Dr. Peet under ether anesthesia and the sensory root of the gasserian divided. The motor root was isolated and was not injured. The patient was discharged cured, seven days after operation. Pain was absent and complete anesthesia of the left side of the face was present.

Subsequent history. The patient went without protection to the left eye and developed a keratitis for which she returned to the Ophthalmologic Department. The pain has not returned.

Case VIII. No. 8300. Mrs. M. R., age 57. Housewife. Family and personal history negative. Present illness began thirty-five years ago, at the age of 22 with sharp shooting pains in the left side of face. These pains have appeared at varying intervals and for periods as long as two years have been very frequent. For several years the pain had been present every day and finally became almost constant, so three years ago the inferior maxillary nerve was avulsed external to the foramen ovale through the Kochler incision. This gave complete relief for one year, when the symptoms returned with even greater severity. All the teeth were removed several years ago without relief and alcohol injections have not stopped the pains. All branches of the fifth nerve are now involved, the lower two being the most seriously affected. The pain is practically constant and has required the continued use of morphine. This drug now gives only slight relief as considerable tolerance has been acquired.

Physical examination shows considerable emaciation. Neurologic examination is negative except for complete paralysis of the motor nerve of the fifth on the left side, the result of the previous operation. No anesthesia from the previous operation.

September 10, 1917, operated upon by Dr. Peet under ether anesthesia. The sensory and motor roots of the gasserian ganglion were divided on the left side. The motor root could not be easily distinguished from the sensory, and inasmuch as motor paralysis of three years duration was present, no serious attempt was made to preserve it. After operation, complete anesthesia of the area supplied by the left fifth nerve was present. Pain was entirely relieved. The patient was discharged cured, ten days after operation.

Subsequent history. The patient was warned to protect the left eye, but went about without protection of any kind. A severe conjunctivitis developed which was untreated, and later a corneal ulcer appeared, when she returned to the Hospital and has been treated in the Ophthalmologic Department.

Case IX. No. 9009. Mr. T. W. Farmer. Age 78. Family and personal history negative. Present illness began five years ago during the winter. He had sharp shooting pains in the region of the right eyebrow which appeared at frequent intervals for one year. The pain then spread to the right side of the nose and gradually increased in severity. One year later the pain included the entire area of distribution of the upper two branches of the fifth nerve on the right side. The paroxysms have increased steadily, both in number and intensity. At the present time they are almost continuous. Attacks are brought on by the slightest touch to right side of face. Eating does not bring on a paroxysm unless the face is accidentally touched. Talking seems to initiate an attack at times. Most of the paroxysms occur spontaneously. The right side of the face is very sensitive. He says he has not lost weight, but has been unable to sleep for more than an hour or two at a time. Physical examination. Low, blowing, systolic murmur heard over precardial area and transmitted to axilla. General condition good, considering his age. Blood pressure, systolic 100, diastolic 70. Wassermann negative. Neurologic examination negative. Patient was transferred almost immediately from the Neurologic Clinic because of the excruciating pain he was suffering.

November 3, 1917, operated upon by Dr. Peet under ether anesthesia. Sensory root of the gasserian ganglion was divided without injury to the motor root. The patient left the operating table in good condition. Six hours later the patient was hungry and ate a light supper. Examination on the third day after the operation showed complete anesthesia of the right side of the face with no motor paralysis. He was in excellent condition, entirely free from pain, and was ordered up in wheel chair the next day. However, during the night he got out of bed and fell to the floor, striking the head. When found he was in deep coma which lasted for twenty minutes. Following this he slightly recovered, talked in an irrational manner, but could not be aroused to answer questions. He never regained consciousness and the following day was in coma again, remaining so until death on the sixth day after operation. Autopsy showed extensive signs of concussion. Hemorrhages were found on both sides of the cerebral cortex, at the base on both sides, within the occipital lobe on the right, and deep in the cerebellum on the left. These last were of the multiple pin point type. This cannot be considered as an operative death. The history given by relatives showed that the patient was in the habit of getting out of bed at night and had fallen several times before.

Discussion. The eye changes which occurred in our patients were all due with one possible exception to carelessness on the part of the patients themselves. All patients after operations for tic douloureux are warned to protect the eyes by wearing a pair of automobile goggles, a Buller shield, or similar device. The eye is usually covered by a Buller shield immediately after operation to protect the cornea from dust or other foreign bodies and to prevent evaporation and drying of the conjunctiva. The eye is washed daily or oftener with boric solution and the patients are requested to do the same on their return home. They are also instructed to go to a competent ophthalmologist as soon as any redness is seen in the eye, and to notice any changes in the conjunctiva since the subjective symptoms will be absent due to the anesthesia. However, several of our patients, feeling so well, and being entirely relieved of pain, have not taken the necessary precautions to protect their eyes and have developed more or less serious complications.

Only one patient developed lagophthalmos. This was undoubtedly due to pressure or stretching of the branch of the facial nerve which supplies the orbicularis palpebrarum, by the retractor. The position of the "question mark" incision precludes any possibility of division of the upper branches of the facial nerve. A paralysis such as this patient had would probably be only temporary but considerable damage could result to the eye in the mean time, since the cornea would not be frequently washed off by closing of the eyelids.

Paralysis of the motor root of the fifth nerve was present in one of our cases, the result of a Kocher operation on the inferior maxillary nerve. Since paralysis already existed, a serious attempt to separate the motor root from the sensory was not made. Excluding this case, the motor root was isolated and not divided in five of the eight sections of the sensory root, or with the case included, a ratio of five uninjured to four divided. Division of the motor root in the Spiller-Frazier operation does not necessarily mean permanent paralysis of masticatory muscles, since several examples of regeneration of this nerve have been reported. No such regeneration is possible when the entire ganglion is removed. The patients however complain very little of this paralysis. An interesting auditory condition was discovered after the intracranial operation. This consisted in a transitory bone conduction deafness of slight degree. The whispered word was heard normally.

It is evident from a perusal of the histories of the cases in this series that peripheral operations are only palliative. Temporary relief, sometimes lasted for a few weeks only, in a few cases relief was experienced for several months, but in every case the paroxysms re-

turned, and generally in greater frequency and severity. Alcoholic injections gave the shortest relief and seldom were of benefit when repeated. Avulsions of the nerve naturally were superior to the injections, but were never permanent in their results. The facial nerve had been injured during the avulsion of the inferior maxillary branch in the ramus of the jaw in two cases seen. This could not happen in the operation for section of the sensory root as practiced here.

*Conclusions.*—Tic douloureux or trigeminal neuralgia must be sharply distinguished from the ordinary forms, since the latter are amenable to simple measures and do not require the drastic procedures which are necessary for permanent relief in the major form.

The pathology of trigeminal neuralgia is obscure. The prevailing opinion at present is of an ascending neuritis of unknown etiology which finally invades the gasserian ganglion. The removal of sound teeth in the hopes of relieving the attacks of pain is absolutely unjustifiable.

Surgical treatment should alone be considered in typical tic douloureux. Nine methods are available, but from the history of the cases in this series, as well as from the experience of other surgeons it is evident that all extracranial operations are only palliative and simply postpone the inevitable intracranial procedure.

The choice then lies between section of the branches distal to the ganglion) only applicable in the lower two divisions), removal of lower part of ganglion, excision of ganglion in toto, and section of the sensory root. The latter is undoubtedly the preferable method, as it is performed with the minimum of complications and permanent relief from pain can be absolutely assured.

Ten intracranial operations were performed in this series. In nine cases the sensory root of the gasserian ganglion was divided, and in one the ganglion was removed in toto. All have had complete relief with no return of the paroxysms.

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DISCUSSION.

Dr. Carl D. Camp: I think that perhaps in discussing the subject of the treatment of trifacial neuralgia we should remember a point which we are very likely to forget, and that is that neuralgia of the face or elsewhere is probably only a symptom. It is true that a certain number of older writers have made a distinction between tic douloureux and trifacial neuralgia and they have considered that tic douloureux is a disease and trifacial neuralgia is a symptom. I am unable to make a differential diagnosis between the two. I believe that in every case the diagnosis of neuralgia is as unsatisfactory as the diagnosis of headache or indigestion. Thesefore the first step in the treatment should be the consideration of what is really wrong with the patient. Of course, in these patients the pain is frequently so severe that one hates to spend the time which may be wasted in putting them through a long series of tests to find out what is the real trouble.

Dr. Peet has said that the alcoholic injection of the peripheral nerve is very often satisfactory, temporarily. I believe that it is a good thing to try at first in every case, especially if only one branch is affected. It is such a simple operation and it produces complete and immediate relief. If it only lasts for a few months there is that much gained at any rate. I have seen cases where to the best of my knowledge and belief a superficial injection has resulted in permanent relief. Dr. Peet spoke of the injection of alcohol into the gasserian ganglion. I gathered that he was possibly a little incredulous. As some of you know, I have done it myself on the living subject. While it is not extremely easy, still it is comparatively easy.

It is done without anesthetic of any kind. The needle is inserted beneath the zygoma and just as near to the depression of the coronoid notch as you can get it. Then, if you have calculated your direction right, it passes directly back into the foramen ovale. In the literature on the subject there is a considerable number of papers by men who have injected only the second and third divisions of the fifth nerve and believed that they had injected the ganglion. At least they have obtained only an analgesia of the cheek. In my own cases I was able to get a total anesthesia of the supraorbital branch and I therefore feel perfectly sure that the needle was in the gasserian ganglion. I gave it up after three cases, not because of unfavorable results, because the patients are still relieved, but thinking the matter over, I came to the conclusion that it was only a question of time until I should kill somebody that way and so I decided to leave the job to the surgeon. It is not difficult but the surrounding structures in the neighborhood of the foramen ovale and just inside are rather important and I believe that the open operation, either gasserectomy or section of the sensory root, is the safer in most cases and this in spite of the statistics to the contrary. I would advise the operation of injection of the ganglion provided there

was some serious surgical objection to the open operation. If the patient was a nephritic or cardiac case who could not take an anesthetic, or for some reason would not have an open operation, and preferred to take the risk of a gasserian injection, I think it would be allowable. Since publishing my injection paper I received a number of letters from patients who wanted to have their ganglia injected, but my replies seemed to dampen their ardor.

Dr. Avery D. Prangen: We have been very much interested in Dr. Peet's series of cases, not only from the ophthalmologic standpoint, but also neurologically. The manifestations of complications in the eye may be divided into three general heads, a lagophthalmos or inability to close the eye, ptosis or inability to open the eye, and corneal pathology. Lagophthalmos, although potentially a serious affair, in this series of cases is only transient and of very little importance. It seems to come on within a very short time after the operation, either the same day or within a day or two. The explanation seems to be rather difficult. The first and more probable possibility is that of pressure from hemorrhage, causing a blocking and temporary paralysis. Inasmuch as the exit of the seventh nerve is in rather close proximity to the field of operation, and also close to the gasserian ganglion, trauma at the time of operation either to the nerve itself in the skull or the branch going to the orbicularis on the face might explain the result, although you would expect a permanent paralysis of the muscle. Indirectly it might occur through the various connections of the facial and the fifth nerve, for instance, through the greater petrosal nerve or some of the more peripheral connections. In the treatment of the lagophthalmos we have put on a Buller's shield and kept the eye clean.

The ptosis is probably of the same general nature as the lagophthalmos.

The corneal pathology is much more serious, inasmuch as it involves the vision of the eye on the side of the operation. It has manifested itself either as a keratitis, an indolent neuroparalytic ulcer or a very virulent infected ulcer. The corneal pathology occurs about the second or third day or may be delayed a week or more. It gives a definite clinical picture. The cornea becomes hazy in the center, the epithelium is stippled and there develops an ulcer in the center. As to the etiology, from our standpoint, it is a very interesting problem. Section of the fifth nerve seems to bring on a trophic nutritional disturbance in the cornea, particularly in the epithelium. The latter seems to lose its normal resistance so that it is more subject to dessication and trauma. Experiments in animals after section of the nerve have brought on keratitis with ulceration. It has been argued that this pathology results from the insensitive cornea being exposed with resulting trauma and infection. However, the condition has developed where the cornea has been protected. So there is probably a trophic nutritional disturbance as the primary cause. Secondarily, there is a decrease in the lachrymal secretion. The epithelium becomes pitted and shows a rapid tendency to slough. So it would seem that the cornea needs more moisture and due to the decrease in the lachrymal secretion it receives not even a normal amount. As another contributory cause we have the insensitive cornea which has lost its normal protective mechanism by stimuli from the lids, conjunctivae and the cornea itself.

The treatment of these cases would seem to divide itself into prophylactic and local. Another common procedure in the course of an operation is to test the corneal reflex as a judge of the depth of anesthesia. The repeated testing of a cornea which is insensitive is bad practice. Second, put on a Buller's shield when the patient leaves the operating room. Then instruct the patient that his cornea must be protected by the senses of sight and hearing. Also have the patient wear protecting lenses to avoid foreign bodies and trauma. When the patient leaves the hospital instruct him to report for treatment upon the appearance of any redness in the eye. When the patient goes to bed at night, put a pad over the eye until he gets up in the morning. These measures would help to avoid the initial trauma. Locally, treat as a corneal ulcer with a pad, atropine and stimulating applications. As to the prognosis, these eye complications are bad as far as acute central vision is concerned, because they always leave a dense central opacity in the cornea. As far as saving the globe and peripheral vision is concerned, the prognosis is good.

Dr. Albert C. Furstenberg: These cases have been interesting to us from the standpoint of differential diagnosis and the functional effects upon the ear. Two cases which had been sent to the Hospital for trifacial neuralgia were referred to our department and found to be suffering from chronic maxillary sinusitis. Both cases were operated upon with complete and permanent relief of the pain.

Following operation the patients have shown very little deafness for ordinary conversational voice but a decrease in the bone conduction and a lowering of the high limit when tested with the tuning forks. These changes are characteristic of slight nerve deafness and are probably the result of traumatism to the eighth nerve during operation. In the cases examined, in from a month to six weeks following the operation the function of the eighth nerve has been almost completely restored to normal.

Dr. Max Peet: First I wish to explain to Dr. Camp that I am not skeptical as to his ability to inject the gasserian ganglion. I do not, however, consider it a safe procedure. All of our cases were warned about the eye and some of them were told before operation that there was a possibility that they might have trouble with the eye which might mean enucleation. The patients all said they would rather lose the eye than have the pain. Two or three were extremely insistent upon operation immediately. They were in poor condition but they said that if they were willing to take the chance, we should be. They suffered so severely that the paralysis of the masticatory muscles and trouble with the eye are completely outweighed by the relief from pain.

# GASTRIC DISTURBANCES AS A PART OF CENTRAL NERVOUS SYSTEM SYPHILIS.

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I want to report the histories of two cases of gastrointestinal disease occurring as a part of cerebrospinal syphilis. My object in bringing these cases before you is to emphasize certain outstanding features of this condition, features which are described in the literature, but which are liable to be overlooked by the practitioner. These features are:

First, severe gastrointestinal disturbances in cerebrospinal lues are frequent.

Second, gastrointestinal disturbances may form the chief complaint in this disease.

Third, symptoms suggesting cerebrospinal syphilis may be vague or entirely absent.

Fourth, the gastric disturbances may occur so early in cerebrospinal lues that none of the older signs of the latter disease may as yet be present and the correct diagnosis may depend entirely upon the examination of the cerebrospinal fluid.

The first case is a man of 29 who came here on the 5th of November complaining of abdominal pain somewhat more marked in the upper abdomen. The family history is of no consequence. There is a record of two living children. His wife had no miscarriages. He had gonorrhea at 18 accompanied by soft chancres. These were not followed by secondary signs of lues. He had another attack of gonorrhea in May, 1917, which has since disappeared. He had an abscess on the tibia eleven years ago which took a very long time to heal.

The present illness began in March, 1912, and came on suddenly with very severe sharp pain in the left upper quadrant of the abdomen beneath the ribs. This pain was relieved by vomiting, but shortly returned. He had an operation for gallstones and later a second operation for appendicitis. Three months after the second operation he returned to work but worked only three weeks when the pains returned. This time vomiting did not relieve him, in fact, he became worse. He was given morphia by his doctor. He had a third operation for adhesions followed by only temporary relief. He had a fourth operation in October, 1914, when a gastroenterostomy was performed. He was advised to have a fifth operation. In some of these attacks he has vomited bright red, sometimes dark blood. He has also had numerous hemorrhages from the bowel. His attacks are quite irregular. His last one started the 26th of September and he came here the 5th of November. The pain usually comes on a few minutes after meals. It is severe, cutting, gnawing, and is sometimes relieved by vomiting and sometimes not.

In his physical examination the things of consequence are an old scar on the left tibia. The pupils are slightly irregular, one pupil being larger than the other. They both react to light and to accommodation. The extraocular movements are normal. The rest of the examination is negative except for a palpable spleen. His abdomen is soft; there is no tenderness, no rigidity, and no masses. There is slight enlargement of the inguinal glands. The biceps and triceps reflexes are diminished but present. The patellar and Achilles jerks are obtained with reenforcement.

So the physical examination shows this irregularity of the pupils and sluggish knee jerks. There is no Romberg and there are no sensory disturbances. The X-ray report favors an appendicitis, but no diagnosis is made. The cerebrospinal fluid showed fifty cells with albumin and globulin present. The Wassermann on the blood is negative. On the cerebrospinal fluid it is markedly positive. He was treated by intraspinal salvarsan. As a result of that his gastrointestinal symptoms have entirely disappeared. He gained about forty pounds, returned to his work and has felt entirely well, athough his gastric disturbances started over four years ago and he has had four abdominal operations.

The second case is a woman of 39 who came here on the 6th of November complaining of stomach trouble. There is nothing of importance in her family history. Her first husband was apparently a syphilitic. She married again. The second husband is living and well. In the winter of 1914 the patient began to lose strength and about ten pounds in weight. Her appetite was poor and she was constipated. In October, 1915, she was seized by a gnawing steady pain in the left upper quadrant which was relieved by eating and taking soda. The pain would return two or three hours after a meal. After three or four weeks she felt entirely well except for slight irregular pain. She continued this way until October, 1916, when she had another severe attack lasting five or six weeks accompanied by nausea and vomiting. October, 1917, she had another attack. In her final attack just before entrance the condition was more severe and she had very severe hem-

orrhages. This history, of course, suggests peptic ulcer.

Examination shows both pupils reacting to light and to accommodation. The left pupil is distinctly irregular. There is no cervical glandular enlargement. The abdomen is level. The liver and spleen are not felt. There is moderate tenderness over the epigastrium and right upper quadrant. The biceps and triceps and patellar and Achilles jerks are normal. There is no Romberg sign.

So the only thing suggesting lues is the irregularity of the left pupil. The Wassermann on the cerebrospinal fluid is ++++. One specimen of blood was ++, another +—. She was transferred to the Department of Dermatology and has had intraspinal treatment. For the last two weeks she has had no gastrointestinal disturbance.

In addition to these two cases we have had three others in this Clinic since the first of October. All of these people came here because of the gastric disturbance. In none of them had the correct diagnosis been made prior to the entrance into the Hospital, this in spite of the fact that two of the cases were actually advanced tabetics.

DISCUSSION.

Dr. Joseph A. Elliott: We have seen in the last year or two many cases of tabes with gastric crises. Some of these have responded very readily to treatment, while others have been rather refractory. Undoubtedly gastic crisis is the most common gastric disturbance associated with tabes. There have been other gastropathies described, such as perforating ulcers, active gummatous lesions and symptoms referable to gastroptosis and enteroptosis. The perforating ulcers are similar to the ones occasionally seen on the feet of tabetics. Bloody vomitus or hemorrhage suggests the presence of such lesions. There is every reason to believe that active gummatous lesions may be found in tabetics as gummata of the skin are sometimes seen in tabetics.

In recent years syphilis of the stomach has attracted the attention of clinicians, who have recognized three distinct forms, viz; the isolated ulcer, multiple ulcers, and a diffuse fibrosis. The symptoms from these lesions are the same as with the peptic ulcer, with the exception that the pain is not relieved by the administration of alkalies. Another point which has been laid stress on by some observers is the low acidity found in syphilis as compared with a very high acidity in peptic ulcer. In Eustermann's twenty-three cases he found an average acidity of 26, and in a few cases found an anacidity. The more advanced gummatous lesions of the stomach resemble very closely malignancy. The loss of weight, however, which is rather marked is not followed by the great loss in strength which occurs in malignancy. All of these cases, however, respond very nicely to specific treatment. After the lesion has entirely disappeared it is sometimes necessary to have the patient operated upon for the resulting constrictions.

Outside the gummatous lesions and the ones previously described there are relatively few syphilides of the stomach. Secondary lesions have been described by Virchow who claims that these lesions occur at the time of the general dessimination of the spirochetes into the blood stream. It is generally conceded at present that the disease gains entrance into the nervous system at that time, so it is reasonable to believe that the different viscera may be likewise affected. Syphilis of the stomach is perhaps more common than we are led to believe and it will take further study and observations to confirm this point.

Dr. Mark Marshall: It is very interesting, of course, to know whether or not there was some local lesion in the cases reported by Dr. Newburgh, something in addition to a central nervous lesion with a secondary manifestation upon the part of the stomach. This problem is met with in every case. I have in mind two private patients who happened to come to me at the same time a few years ago. Both were diagnosed chronic appendicitis and operated upon by the same surgeon. Each had complained of stomach symptoms regarded as due to a chronic appendix. Neither of them improved and it was found in one case that the Wassermann was ++++ and the pupils irregular and the man went on to an advanced stage of general paresis and finally died. His stomach findings were compatible with the symptoms which he showed. The stomach was enlarged and dilated and he had a low degree of stasis and occasional vomiting. Strange to say, however, his stomach symptoms improved remarkably with salvarsan, mercury and potassium iodide. In the other case the stomach findings were of no consequence. The laboratory findings were not strongly positive for syphilis but all of his signs and symptoms, and particularly his mental state was characteristic of an early general paralysis and he had to be committed on account of his mental changes. He was not improved by antisyphilitic treatment.

There is an interesting case which we had in the Medical Clinic. This was a patient with stomach symptoms in which cancer was suspected and I believe the patient was explored. Afterwards, she was found to have syphilis and was treated thoroughly and improved, but came back some months later with an inoperable carcinoma. The mere fact that the patient improves under antisyphilitic treatment does not necessarily prove that the gastric condition was gastric syphilis.

Dr. Harold DeB. Barss: The question which occurs to me is this: If the symptoms exhibited by these two patients are frankly and only due to central nervous system lues, I think we shall have to revise our mental picture of gastric crises. These attacks are typical of definite lesions in the gastrointestinal tract that surgeons are accustomed to treat without evidence of luetic infection. If these are symptoms of central nervous system lues apart from local conditions, then the surgeon will have to revise his mental picture and make more careful routine examination of all his cases to rule out neurologic findings. One interesting question is whether central nervous system lues without a local lesion will give the ordinary X-ray picture which

we find in local lesions. I would like to ask Dr. Van Zwaluwenburg whether central nervous system lues without local lesions gives a typical picture.

Dr. Quinter O. Gilbert: Since these cases were brought up chiefly because of the problems of diagnosis. there is one point in diagnosing early tabes to which I wish to call attention because so often the test is not correctly done. The point in question is the light reflex for the eye. Many people confuse the consensual and direct light reflx and do not obtain the loss of light reflex, because the patient had not fixed for accommodation. One should test always for both the consensual and direct reflexes. because in a large number of patients the consensual reflex is lost before the direct. Again this will act as a check on the direct. I have seen the "fixed pupil" called normal, because the accommodation reflex was so great, when the patient had not properly fixed for distance. The pocket flash light which is commonly used now for testing the eyes, may lead to the confusion above mentioned. Invariably if the light is held in the field of vision and flashed, the patient accommodates for the near object. The proper way to use the light is to have the patient fix on a distant object and then play a beam of light into the pupil without making any other manoeuver which will attract the attention, thus calling into play the accommodation for distance.

Dr. James G. Van Zwaluwenburg: I am interested in this subject and have usually met with chagrin. It is impossible here to cover the whole subject of gastrointestinal syphilis which Dr. Elliott has well considered. The gummatous and scar tissue lesions and the contractures following the gumma, are usually mistaken for carcinoma radiographically and most of them have not been recognized before operation. Several of them have been closed as being inoperable carcinomata and the true condition has not been discovered until the patients failed to die as per schedule and following antisyphilitic treatment have improved, although it must be said that the stomach never returns to its normal shape.

As for the occurrence of an actual ulcer, we must concede that it is possible, and probably does occur. Yet the operative reports on syphilitics which have been explored for ulcer have usually failed to reveal ulcer. We have three or four cases in this Hospital which I remember. As for the other symptoms, I think we are as much at a loss from the radiographic as we are from the clinical point of view. To a large extent our conclusions are drawn from altered motility of the gastrointestinal tract. We believe that these follow alterations in the normal reflexes of the gastrointestinal tract, all of which we do not yet fully understand. These reflexes are naturally under central nervous control and naturally any central nervous disease may modify these reflexes. My own experience has been rather disappointing in these cases. Twice I have been saved from making a fatal error. Two patients with a history of peptic ulcer came for examination showing fairly typical pictures of ulcer radiographically. When I examined them in the dark room they complained of being dizzy and one of them nearly fell. On examination I found a definite Romberg. Such evidence was not purely radiographic and waiving the usual rule on the evidence of the Romberg, I did *not* make the diagnosis of peptic ulcer. The remainder of the cases have looked very much like appendicitis. In most instances they have not been very convincing. I think that is about our attitude in the matter. We concede that syphilis may simulate practically any condition radiographically and that we commonly find any of the surgical conditions simulated. I know of no way to differentiate.

Dr. Barss: If these patients don't improve under antisyphilitic treatment, perhaps they have appendicitis.

Dr. Van Zwaluwenburg: On the other hand, with the altered central control of reflexes I doubt whether we can draw any conclusions.

Dr. Barss: In the first case it is interesting to note that after four operations he can feel perfectly well even with antisyphilitic treatment.

Dr. Van Zwaluwenburg: A radiographic diagnosis of appendicitis does not mean that the appendix is still there. What we recognize are the changes consecutive to the appendicitis or an operation.

Dr. Newburgh (Closing the discussion): Dr. Barss's remarks about gastric crises bring up the important point that syphilis causes gastrointestinal disturbances which are not typical gastric crises. We have heretofore waited for the typical picture before making the diagnosis. Perhaps half of these cases do not have the typical crises. They may simulate any of the formal varieties of the gastrointestinal diseases. The history of the attack is of no help unless it is a history of a crisis. The history may suggest hyperacidity or ulcer or carcinoma or anything. The important point is to remember that a number of persons complaining of a variety of gastric disturbances may be suffering from lues of the central nervous system and the final diagnosis can only be made by keeping that in mind in every case and excluding central nervous system involvement in every case of gastrointestinal disease.

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## RECOVERY OF A CASE OF PURULENT MENINGITIS COMPLICATING MASTOIDITIS.

D. O. Walthall, M.D.

(From the Pediatric Clinic, University Hospital, Ann Arbor, Michigan).

The rarity of recovery from otitic meningitis was the incentive for reporting this case and discussing the condition. The mortality of this disease is over 90 per cent and until 1893 when Macewan reported six recoveries out of twelve cases, no serious attempt had been made to treat suppurative otitic meningitis. Meningitis follows chronic otitis twice as frequently as it does the acute form. In the acute otititis the fulminating and explosive type of meningitis occurs, while in chronic otitis the onset is insidious and it may be from a few days to several months before the meningitis is clearly defined. Usually when the symptoms are serious enough to call for operation the chances of recovery are gone. Therefore mastoidectomy and decompression should be regarded as exploratory oper-

tions and resorted to with the same freedom as in doubtful appendicitis and other abdominal riddles. The surgical treatment then becomes prophylactic.

CASE I. Small girl, age 7 years, had earache and discharge from the ear in January, 1917. She had good health until August 25th when she developed pain in the right side of the head and a sensation of something breaking in the ear but no discharge. She entered the Hospital September 15th with the same symptoms. Paracentesis done on the right ear and September 18th, a right mastoid operation. She had an uneventful convalescence and went home September 29th against advice. On October 13th she developed a temperature and felt dizzy. She returned to the Hospital on October 16th and was seen in consultation on October 18th because of temperature. A history of vomiting during the night and morning was obtained. She had a slight headache and did not want to be disturbed.

*Examination.*—Cheeks flushed and dark circles under her eyes; lips dry; cervical glands enlarged; head slightly drawn back. Patient more comfortable when lying on the side. Neck very stiff. No neck sign (Brudzinski). Pain in the right ear. Mastoid wound not filled in but clean. Eyes; lateral nystagmus, slight diplopia. Pupils dilated but react. Eyegrounds not done. Knee jerks lively. Babinski suggestive at times. Kernig slightly positive. General physical examination normal. White blood count 19,000.

The mastoid wound still did not fill in and remained the same with practically no discharge. On the morning of November 27th the dressing was changed and was clean. During the early evening the nurse noticed a very foul odor in the mastoid region. The dressings were changed and were found saturated with foul pus. A sterile saline irrigation was given. The mastoid wound is being dressed every day at which time there is a large evacuation of foul pus which saturates the dressings. There is no temperature and the patient is up and playing about the ward.

The following case although it cannot be included under the same heading deserves brief mention, owing to its resemblance to the above.

CASE II. Female, age 42, married. The family history, past history and marital history have no bearing. On November 15th she was suddenly taken with a sharp pain in the head. The pain was so great that the patient became practically irrational from the first and was confined to bed. On November 7th the right ear drum perforated and discharged. At this time the pupils were widely dilated. On November 10th vomiting began. During all this time the patient's temperature ranged from 102 to 105 degrees. On November 11th she became unconscious. November 14th she entered the Contagious Hospital under a mistaken diagnosis. The temperature was 104 degrees, pulse 108, respiration 36.

*Examination.*—Patient unconscious; head drawn back and to the left; definite stiff neck and neck sign. Very irritable during the examination and cries out each time she is touched or moved. Pupils sluggish to light. Ears; left drum normal; right drum bulging and red. Paracentesis freed a large amount of thick pus. Reflexes all much exaggerated; definite and marked double Kernig. Tache cerebral present. Heart, lungs, and abdomen normal. White blood count 35,000. Lumbar puncture; 30 c. c. of turbid fluid under much increased pressure came away. Total cells 1000 per c. c. Polymorphonuclears 98 per cent. Lymphocytes 2 per cent. On standing a few minutes, coagulation occurred. Smear showed Gram positive diplococci-pneumococcus. Group 3 by agglutination. Repeated lumbar punctures were done which showed a steady increase in cells and organisms. The patient did not rally but

CHART OF PUNCTURES AND TEMPERATURES.

| Date | Temp. | Amount fluid c. c. | Turbidity | Total cells | Differential Polys. | Differential Lymp. | Organisms |
|---|---|---|---|---|---|---|---|
| 10–18 | 103.6 | 30 | Pus | 1800 | 60 | 40 | Gram + |
| 10–19 | 101.6 | 15-20 | Cloudy | 1300 | 69 | 31 | No definite organisms |
| 10–20 | 105 | 30 | Pus Pressure + | 5460 | 80 | 20 | Rare cocci with M. B. |
| 10–21 | 103.6 | 10 | Cloudy | 1100 | 60 | 40 | Stained smear 0 |
| 10–23 | 100.8 | 10–15 | Cloudy | 570 | 35 | 65 | Stained smear 0 |
| 10–24 | 100.4 | 35 | Cloudy Pressure + | 760 | 30 | 70 | Stained smear 0 |
| 10–30 | 98.6 | 20–25 | Opalescent | 160 | 11 | 88 | Stained smear 0 |
| 11–7 | 98.6 | 20–25 | Clear Pressure normal | 30 | All lymph. | | Culture 7 out of 10 tubes + staph. |
| 11–17 | 98.4 | 20–25 | Clear Pressure normal | 40 | All lymph. | | Culture 0 |
| 12–1 | 97.8 | 30-35 | Clear Pressure + | 10 | All lymph. | | Culture +, staph. |

developed rales and pneumonia in the left lower lung and died November 16th.

Post-mortem. "Chronic otitis media on the right side, with complete destruction of the ossicles and purulent infiltration and necrosis of the greater portion of the petrous temporal upon the right. Pneumococcus infection. Acute fibrinopurulent leptomeningitis. Metastatic miliary abscess of the kidney. Acute passive congestion of all organs."

I want to thank Dr. Weller for the autopsy report and Robert Novy for the bacteriologic work.

CONCLUSIONS.

1. Early diagnosis must be made before the textbook symptoms appear.

2. Every mastoid operation should be preceded by a lumbar puncture.

3. Otitic meningitis is a preventable disease made so by prompt and effective prophylaxis through surgical intervention.

DISCUSSION.

Dr. Albert C. Furstenberg: The first case has been of especial interest to us because so far it seems to demonstrate an apparent recovery. When this patient entered the Hospital there were all the characteristic signs and symptoms of suppurating middle ear and mastoid infection with a large subperiosteal abscess. At operation it was discovered that there was a fistulous tract extending from the mastoid process upward and downward deep into the petrous portion of the temporal bone to its roof. At its termination the dura was uncovered and there was found a small area of external pachymeningitis about one centimeter in diameter. The bone was removed until healthy dura surrounded it on all sides. A very extensive mastoid operation was performed, the entire sigmoid sinus was uncovered and all the mastoid cells deep into the temporal bone and the tip were removed. After the operation granulation took place promptly. On the twelfth day after operation, on account of financial reasons, the child was discharged against advice. The ear was practically dry and the tympanic membrane had assumed its normal appearance. About a month later the patient was brought into the Hospital with a history of nausea, vomiting, and dizziness. The patient showed a temperature of 102 degrees. At the time that the pediatricians were called over there were no very characteristic signs of meningitis, the Kernig was not demonstrated positively and the rigidity of the neck might well have been due to the mastoid operation. There was a slight dilatation of the pupils and a temperature. The lumbar puncture, however, showed that the fluid was purulent. At this time it became a question as to whether or not another operation should be performed. All of the otologists who report cures report them after radical procedures with removal of a large part of the petrous portion of the temporal bone exposing the dura and making numerous incisions into the dura for drainage. It did not seem feasible to do this and form new avenues for the entrance of infection. From the signs and symptoms and progress of the disease it would seem quite evident that the pathology was circumscribed and not diffuse. I believe, however, that operation should be instituted in cases of meningitis before the patient becomes in an extreme condition and that a radical operation should be performed to remove the focus of infection. But when dealing with a circumscribed subdural process and where there is good drainage it does not seem advisable to incise extensively the dura and break down the barriers in the subdural space that have already formed.

Dr. Max Peet: Dr. Walthall spoke of the Haynes operation for general meningitis. This operation appears to me to offer the best opportunity for drainage in these cases. And it is by drainage alone that we can hope for a reduction in the mortality. The mastoid operation, extended to expose the dura, gives excellent results in very localized meningitis of otitic origin. But in generalized meningitis, and we are here speaking of those due to such organisms as the staphylococcus, streptococcus, and pneumococcus, not from the diplococcus of Weichselbaum, it is impossible to expose the entire area involved and our only recourse is free drainage which reduces the terrific intracranial pressure, removes an enormous amount of infected and toxic material, and gives the body a chance to combat the disease by natural methods. We believe implicitly in the evacuation of pus and drainage in all other parts of the body, why should not the same procedure be applied to the meninges? The Haynes operation opens the cysterna magna at its most dependent part and allows of the evacuation, by the finger if necessary, of thickened masses of pus, and the continued free drainage of infected cerebrospinal fluid. The operation can be performed in one-half hour and in many cases little or no anesthesia is required. There are no vital structures to be injured until the dura is exposed and the operation can be performed by anyone who has had experience in cerebellar surgery.

I think that repeated lumbar punctures are beneficial, but they are at most palliative, not curative.

I have performed the Haynes operation once, on a case of streptococcus meningitis. The man had been unconscious three days and was sent in with a diagnosis of peritonitis. On opening the cysterna magna great quantities of pus containing large flakes were evacuated. The fluid resembled that obtained on opening an empyema of several days duration.

A half hour after operation he had regained consciousness and asked for a glass of water. He died the next day.

The value of any operation such as the Haynes depends upon how soon it is performed after the onset of the disease. I personally have never seen a case of purulent meningitis recover other than those due to the meningococcus.

Dr. Walthall: How many Haynes operation cases have recovered?

Dr. Peet: At the time I operated, the Haynes operation had been performed about fourteen times. All these patients had died. But no case so far as I know has been properly drained by the Haynes operation early in the disease. I think, however, that any case of purulent meningitis when the condition has spread to the ventricles is sure to die unless operated upon, and since it is otherwise a hopeless condition, there is not reason why these patients should not be given the one chance which is open to them.

# TRANSACTIONS

OF THE

# Clinical Society of the University of Michigan

## Stated Meeting, January 9, 1918

**The President, JAMES G. VAN ZWALUWENBURG, M.D., in the Chair**
**Reported by REUBEN PETERSON, M.D., Secretary**

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## REPORT OF AN UNUSUAL CASE OF RENAL CALCULUS.

ROBERT H. BAKER, M.D.

(From the Surgical Clinic, University Hospital, Ann Arbor, Michigan).

The case which I have to report is that of a Greek, 34 years of age, who came to the Hospital primarily for Wassermann examination and treatment of syphilis. His family history is unimportant except that he came from a distinctly tuberculous family. His chief complaint in our Clinic was pain in the lumbar region extending across the back from the midline around to the left side, more severe on the left side. His past history shows a chronic appendicitis from youth for which he was operated upon. He came to America in 1907 and contracted gonorrhea and syphilis. He was treated for six weeks and discharged cured. In 1912 he had an acute attack of appendicitis, was operated upon and discharged in twelve days. He has had acute rheumatism in the left leg with stiffness and pain extending to the foot. He was confined to bed for six months in 1913 with this complaint.

His present illness began in November, 1916, with vague pains low down in the back. His work kept him on his feet and while standing his pain was more constant and relieved by sitting or lying down. The condition became progressively worse until February, 1917, when he began to notice frequent urination. This latter symptom became somewhat painful with pain following the micturition. He noticed his urine was cloudy. In March his doctor gave him internal medication without relief. In May, 1917, he began to feel weak all over, and could not sleep nights on account of a backache. He had lost ten pounds in weight. In July he quit work, the symptoms returning with some burning urination. He came to the Hospital in November and was treated in the Department of Dermatology for three weeks previously to entering our Clinic.

His physical examination was entirely negative. At our first examination we noted that he had no definite area of tenderness on either side, although he complained of dull pain on the left. We noted later that nothing could be found on the right side. The attack which caused him to be referred to us came on in the night following a salvarsan injection. This was a definite attack of renal colic on the left side. He gave no history of passing a stone, or of bright or dark blood. His tenderness and pain were so unimportant that we were unable to get a history of previous backache. He denied ever having had anything except a dull backache, which had given him no trouble. He denied all history of any distress upon the right side.

December 6th, I examined him and found a bladder mucosa which was normal except for slight duskiness over the trigone. Both ureters were catheterized with ease, but no specimen was obtained from the right side. The left side revealed urine slightly under pressure. At this examination his bladder urine was alkaline, containing albumen, pus, and some blood. Examination of his left kidney urine was absolutely negative, with no albumen, pus, or blood. The left kidney urine was acid. The patient was taken to the X-ray room for pyleogram, where the left kidney pelvis was injected, and nothing unusual found except that the catheter was completely turned over in the pelvis, indicating a large pelvis. The catheter on the right side reached a point which should be the kidney pelvis, but we were unable to force any thorium into the kidney. The X-ray report from this examination stimulated further study. The picture was so unusual that my first thought was that this patient had had a previous barium meal, and that the shadow on the right side was that of retained barium in the bowels. The unusual configuration suggests folds in the colon. The diagnosis was calcification of an old diseased kidney. In an attempt to verify this, a wax tipped catheter which had previously been examined microscopically for scratches, was passed up the right ureter. The waxed tipped catheter was passed into the bladder before the cystoscope to obviate any scratching at that time. Upon manipulation of the catheter after reaching the pelvis of the kidney, and then withdrawing the cystoscope before we withdrew the catheter, we were able to demonstrate definite scratches upon the waxed tip. This examination was repeated three days later to verify the first finding. There was no doubt then that the patient had a stone in the pelvis of the kidney which was causing the trouble.

The unusual features of this case were the size of the stone, the fact that the patient had

had practically no symptoms, and that the symptoms which he had were all left sided.

The one thing which called our attention to his condition was the acute renal colic following his salvarsan injection. In this attack, the pain was very severe, and would have been interpreted under ordinary circumstances as a colic caused by the passage of a small stone down the ureter. I have attempted to investigate the cause of referred pain from one kidney to the other. It is not uncommon to have such referred pain in hydronephrosis, or ptosis. I know of no physiologic or anatomic explanation of this. I would interpret this case as a result of two factors. Undoubtedly his pain which had existed for at least a year, was due to the gradual formation of this stone upon the right side, for which he had a referred pain on the left side. The acute colic following the salvarsan injection might be interpreted as one of two things. Either the left kidney was taking the strain of his whole excretory function, and was so overpowered by the salvarsan that the hyperemia and congestion caused the pain, or the right kidney might have taken some of the strain, and he felt a renal colic which was referred to the left side.

The patient was advised to have an operation to which he submitted. Instead of finding one large stone, we found a large number of stones, which had so completely displaced the kidney tissue that the kidney had to be removed entirely. It is not uncommon to find stones as large as this, but as you know, the symptoms by which we usually diagnose renal calculus are renal colic, hematuria, and pus in the urine. This man must have had some secretion from the right side, which would explain the pus in the bladder, and the alkalinity of the bladder urine when the urine from the left ureter was acid. The diagnosis was made by the X-ray and the wax tipped catheter.

DISCUSSION.

DR. MARK MARHALL: Is there any record of a case where one kidney has been removed, or is not functionating, in which salvarsan has produced pain from overloading or congestion?

DR. BAKER: I am not prepared to answer that question. That point has interested me particularly. The question has come up as to the further treatment of syphilis in this case, and as to whether it was the salvarsan which caused the colic.

DR. LYLE B. KINGERY: I remember this case quite well because the reaction which followed the first injection was rather unusual. I do not know that I can add anything to the theories of explanation. We know that all the salvarsan of an injection is probably excreted within twenty-four hours following the injection, mostly by way of the kidneys. With this added work thrown upon the kidneys there would necessarily be a congestion. Occasionally we do find slight pains in the small of the back following salvarsan, particularly large doses, which are probably caused by a transitory hyperemia of the kidneys. If the affected kidney in this case were secreting at all, it was without doubt, called upon to do an extra amount of work following the injection, and with this increased strain it is reasonable to suppose that there would be some sort of a reaction such as we had here. I cannot otherwise explain the question of reference of pain from one side to another. I think the future use of salvarsan in the case should rest entirely upon the condition of the kidney. However, provided salvarsan is given cautiously, and governed by the ability of the kidney to take care of it, the question to be decided is whether the benefit is worth the risk. The relation of this injection of salvarsan to the attack itself, I think can be explained no better than as Dr. Baker has already suggested.

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## THE TREATMENT OF CHRONIC CONSTIPATION WITH PSYCHOTHERAPY.

MARK MARSHALL, M.D.

(From the Medical Clinic, University Hospital, Ann Arbor, Michigan).

With no desire to foist upon this Society a tedious discussion of a commonplace subject, but rather to fulfill a certain obligation to Dr. Van Zwaluwenburg, who dictated the assignment, the writer consented to read this paper.

The symptomatic treatment of chronic constipation by the giving of repeated doses of cathartic is such a simple procedure that many laymen regard themselves as competent to manage their own cases and the physician so often finds that this method meets the approval of the patient that little time or effort is expended in seeking a more rational form of therapy. There are, however, a goodly number of patients who have become dissatisfied with the constant use of cathartics, and who demand of the physician a form of treatment that will enable them to discontinue the use of medicine. In such cases the physician usually outlines for the patient an anticonstipation diet, selecting foods which have a large residue, and may even suggest to them the value of regular habits of going to stool. With this advice the patient is left to his own devices and obtains usually only a partial and temporary relief. As a rule he continues to rely at times upon cathartics which practice sooner or later leads him into his old habit of constipation. The management of a case by this method requires one or possibly two visits to the physician's office. The results are pretty

much in proportion to the time spent in obtaining them.

It is the purpose of this paper to present and to emphasize more rational methods of treatment for this very common condition, which in the hands of those who are sufficiently interested, has given remarkable results. It is based upon the very obvious theory that the intestinal tract of the average man will functionate normally, so far as defecation is concerned, if it is not tampered with. This hypothesis might rather be stated as a fact, since it is a fact, but the perverted mind of man has not accepted it as such in formulating his attitude toward the intestinal functions, so it needs must be stated as a theory until such time as accumulated clinical evidences demonstrate it as a fact. Select at random any normal appearing layman, and obtain from him his views concerning his intestinal functions. The chances are, ten to one, he is not satisfied with the natural behaviour of his intestinal tract, however free from symptoms he may be, but thinks he can improve upon nature by taking a purgative occasionally, once a week or perhaps once a month. And how can we blame the poor layman for harboring such a belief, when we realize that every time he ever consulted a physician about a symptom, no matter what the illness or the complaint, the physician gave him a purgative. He naturally comes to look upon the bowels as the hiding place for all disease, and that an occasional sweeping out is the course of wisdom. This tampering with the intestine by the use of cathartics, suppositories and various enemata, is too common to need further comment and frequently marks the beginning of fixed habits of medication. It is a well known fact that there is always a period of constipation following the use of a cathartic, and that with the too frequent use of a cathartic this period of reaction soon becomes the dominant feature.

There is another sort of interference with the bowel movements which, while not so obvious as the preceding, yet nevertheless plays an even greater part in establishing a constipated habit. I refer to the mental attitude of the individual toward his intestinal behaviour, an attitude which manifests itself for the most part in a deeply rooted concern as to whether the bowels are going to move with daily regularity or whether the amount passed is adequate to prevent an autointoxication, a more or less mythical physiologic phenomenon, which by virtue of its apparent plausibility has too frequently served as a cloak to hide our diagnostic shabbiness.

It is a well established fact that even the slightest concern in regard to ones business or professional appointment, will frequently bring about failure of the daily bowel movement. Concern about ones self or ones own health while obviously a far more important factor in establishing such irregularities is usually overlooked by the physician in his analysis of a given case. He recognizes the concern, but regards it, as does the patient, as an effect rather than as a cause of intestinal irregularities. It is a comparatively simple matter to regulate by schedule the physical habits of an individual and thus eliminate many factors productive of habitual constipation, but to regulate his mental habits, is a problem which demands determination, time and infinite patience. It can be done, however, and with such constant and brilliant results that one cannot help regarding most cases of chronic constipation as neurologic conditions, and best treated by psychotherapy.

The general principle involved in the psychotherapeutic management of this condition is not essentially different from that involved in the psychotherapeutic treatment of any functional condition. It presupposes upon the part of the patient a perfect confidence in the physician. After this is attained, the physician has only to suggest the desired result and it comes. This is the underlying principle briefly stated, the detailed application of the principle is somewhat more complicated and will vary a great deal in the hands of different therapeutists and in the management of different cases. No doubt every one in this kind of work developes his own individual technic, but in order to place this presentation on a concrete basis, the writer will outline in detail his own method of procedure. One begins by telling the patient that in order to come to a proper understanding of his condition it will be necessary to make a very thorough examination. As a rational human being, this will appeal to him. One makes the usual physical examination giving special attention to the abdomen. A rectal examination is always made, and if there is any indication for it, a gastroanalysis. The urine and blood are examined routinely. This examination may cover a considerable time during which one purposely avoids treatment of any kind. If, as in most cases, no obvious cause is found for the constipation, one emphatically assures the patient, that if he follows instructions in detail he will never have to take another dose of med-

icine for his bowels as long as he lives. He is told that a thorough examination has failed to elicit any organic basis for his complaint, that his trouble is of a functional nature. Most laymen have a fairly definite understanding of the difference between a functional and an organic condition. He is told that being normal in every respect, his intestinal tract should behave normally and would do so if its activities were not interfered with in any way; that all of ones internal organs are designed to work harmoniously and automatically and unless they are organically diseased they will do so if not meddled with. In the first place they should not be interfered with by the use of cathartics. The patient is then told of the more important source of interference, namely, the effect of efferent impulses arising within the brain, which inhibit the normal relaxation of the anal sphincter and the proper contraction of the rectal muscles. His attention is called to the common experience of failure of defecation as a result of unusual early morning appointments. An appointment at 8:00 A. M., if it is sufficiently unusual, will inhibit the normal act of defecation which otherwise would have occurred at 7:00, the failure not being due to lack of time but to inhibitory influences. The patient in this way is made to realize the importance of such factors and particularly the inhibitory influence exerted by any worry involving the act of defecation itself. These elementary points in physiology are restated in varying terms and supplemented by concrete examples until the patient has a thorough foundation for further instruction. He is then told to free himself from all concern, as to his internal organs, and to place the responsibility of his case in the hands of his physician. He is instructed to go to stool at a certain time every morning within a half hour after his breakfast and to remain there for ten minutes without undue effort to make the bowels move. If they fail to move, he is to completely disregard the fact as the failure cannot possibly do harm. At this juncture one proceeds to explode the autointoxication theory, which in most instances constitutes a strong foundation upon which the patient has built a vast superstructure of therapeutic fallacies. His ideas in this regard are apt to be deeply rooted, since they are based upon statements eminating from scientific sources, and reinforced by the repeated observations of a physician giving a cathartic as the essential part of the treatment of any illness regardless of the nature of the symptoms. To say that one proceeds to explode the patient's theory of autointoxication is really overstating the case, for this is hardly the expression for describing a process so tedious. It often requires a great deal of argument to convince the patient in this matter. Merely to state the fact without convincing him leaves a weak point in the presentation of the case, which to be successful must appeal to the patient as uniformly sound. One can state as a fact that there are healthy individuals whose bowels move but once a week and that no symptoms of intoxication such as headache and lassitude are experienced. On the other hand there are many who will develop a headache before noon if the bowels fail to move in the morning, an interval of time between cause an effect which is entirely too short to admit of such an interpretation. If the bowels fail to move in the morning, the fact is not only to be disregarded by the patient but should there be a tendency for them to move later in the day, this should be disregarded and defecation postponed until the following morning at the proper time. This for the obvious purpose of establishing a regularity to the function. The patient comes to the physician's office every day for the first few days of treatment and reports his condition. These frequent visits are essential for they serve to prevent the patient from assuming any responsibility for his condition. Responsibility leads to concern, doubt and worry, all factors detrimental to the successful management of the case. Should the bowels fail to move for two consecutive days (a rare occurrence, even at the beginning of treatment) the patient is given a simple enema. It is invariably given in the morning at the appointed time. Every day the patient is reassured in regard to his condition. One speaks of the many cases treated without a single failure; of cases much more serious than his own that were readily relieved, etc.

A very practical aid in carrying out this treatment is to arrange for the patient a daily schedule. Particularly a fixed schedule for the early morning hours, one which provides ample time between the hour of rising and the time for going to work. For example, for students with 8:00 o'clock classes, the following schedule has been used.

Rise at 6:30 A. M.; breakfast at 7:00. Between breakfast and 8:00 the patient may read, smoke or do anything he likes except work. The inhibitory influence of early morning work is striking. One patient who was an office worker and who preferred to attend to his toilet after

going to the office, was cured by carrying out the only order that was given him, namely, to assume none of his daily duties until the bowels had moved. When, as occasionally happened, he yielded to the temptation to read his morning mail first, the movement failed. It has occurred to the writer, that the constipation in women which almost invariably has its beginning with the first baby, is far more likely to be due to the irregular life and insistant demands incident to the care of a new baby than to the physical traumatism resulting from the pregnancy.

A detailed report of cases of constipation treated with psychotherapy would not interest you particularly. A brief summary of a few typical cases might be worth while.

A maiden lady, age 56, was referred by Dr. R. B. Canfield June 23, 1913, complaining of occasional cramp-like pains in the abdomen and chronic constipation. She was operated six years before, at which time a fibroid uterus was removed and both ovaries. Two years following the operation, the cramp-like pains began and had been the source of trouble ever since. The pains were described in ways that suggested either the pains of colonic adhesions or possibly the pains which accompany the taking of too much cathartic. July 7th, two weeks after treatment was begun the bowels were moving every other day without assistance, and the pains were less severe. Later the movements occurred daily. Two years after the treatment of her constipation she was operated upon for gallstones. The disturbed conditions incident to the operation and the taking of cathartics while in the hospital caused a return of the bowel trouble, which she assured me she could take care of herself, after returning to normal conditions. She has not been heard from since, so it may be taken for granted she is well.

Case II. A male student, age 22, applied for treatment for constipation, May 11, 1915. For three years he had been subject to severe attacks of constipation. Patient says he does not like to take medicine for the constipation because he believes it makes the condition worse. The first treatment was given three days later. The following morning the bowels moved without difficulty. During the following week the bowels moved every other day. The patient expressed some concern in regard to the infrequent movements. He was told that this could do not harm, and at any time they might begin to move every day. Daily movements began the morning following this suggestion. June 24th, six weeks after treatment was begun, the patient called to tell me of his complete cure, which fact was demonstrated to his satisfaction during the previous week, when he spent four days and nights at a house party, under conditions which previously had never failed to cause trouble.

Case III. A physician, age 40. Had taken cascara every night for four years. He became interested in the method of treatment employed by the writer and applied for treatment. He wished, however, to make an exception to the usual rule, in that he thought it best to continue the cathartic in gradually diminishing doses. This reservation marked a lack of complete confidence in the method, which predicted ultimate failure, if treatment were attempted under such conditions. He was presented with the necessary statistics and arguments to establish complete confidence, and began his treatment under proper conditions. He was cured in a short time. During the past five years the patient has not taken a cathartic.

Case IV. A girl age 24 was referred to Dr. R. B. Canfield July 7th, 1913. Patient had been obstinately constipated for six years. For the past year had to take large doses of castor oil every three or four days, and these gave only ordinary results. The patient was almost totally deaf, so her first therapeutic lesson was given to her in typewritten form. The following morning her bowels moved normally without assistance. Unfortunately the patient left town at this juncture and could not be properly dealt with and in fact received no further psychotherapy. However, a letter received from her three months later, spoke of a marked improvement, though she was still using cathartics, though of a milder sort. This case, while not in any sense cured, was of particular interest in that she showed such a remarkable suggestability, responding as she did to a written suggestion and later maintaining a very definite improvement.

Dr. Curry of a recent graduating class, while yet a student, became interested in the method and undertook the treatment of a very severe case in one of his acquaintances. The case was an obstinate one of six years standing. In six weeks he was entirely cured. Dr. Curry made a most interesting report of the case to the senior class in therapeutics at the time.

Dr. Leslie DeWitt of a previous class, successfully treated a case during his senior year.

About ten years ago, Lyons of Buffalo, reported in the Transactions of the American Society of Physicians, a series of sixty-nine cases treated, with sixty-eight cures. It was this report which stimulated the writer's interest in the method.

DuBois in his book on psychotherapy has a most interesting chapter devoted to gastrointestinal conditions, in which he discusses at length the treatment of constipation. DuBois claims to have had no failures except in cases with a definite organic basis for the trouble.

He recounts an interesting instance in which one of his former pupils, who was very proficient in psychotherapeutic methods, failed in the treatment of a case of constipation, which was manifestly of nervous origin. In order to determine the exact cause of the failure, DuBois asked the physician to relate to him, word for word, his conversation with the patient. The weak point in his argument was found in the following expression. "I am sure I can cure you of your trouble, but if I can't, I know a man who can." DuBois then took the case and within twenty-four hours after the first treatment, the patient began having daily evacuations. Other writers have called attention to the method but nevertheless, it has not come into general use for certain obvious reasons, the first of which is a general lack of interest and confidence among physicians in all psychotherapeutic procedures. The physician also feels that such methods are not only time consuming but are not financially productive. He fails to recognize the fact that a successfully treated patient is willing to pay full value for his advice.

It will be noted that this paper has scrupulously avoided controversial question, such as, what constitutes constipation? May it not be a thing entirely independent of the frequency of defecation? Are there not many individuals who think they are constipated, who only imagine it, and who only have to be convinced of this fallacy, in order to perfect a cure? In other words, what diagnostic standard shall we adopt in defining the condition? The writer has accepted the standard of the patient, which obviously will include varying types of cases under the one heading and might legitimately be regarded therefore, as unscientific. On the other hand, the attempt to classify all cases under the heads of spastic constipation, paralytic constipation and the more fanciful, latent constipation, while it may be very attractive from the standpoint of diagnosis, has been such a failure as a basis for an equally rationalized therapy, that one may be justified in disregarding the whole scheme, even at the risk of being numbered among the unscientific, in order to obtain the maximum number of cures.

DISCUSSION.

Dr. Theophil Klingmann: I was much interested in Dr. Marshall's paper. I take it that most of his cases were without any definite neurologic condition. At least Dr. Marshall didn't mention such conditions. I dare say my experience is very limited in that class of cases.

It has been a great satisfaction to me, however, to observe the results in neurologic cases. Most patients with hysteria subjected to the isolation treatment suffer from constipation. It is usually the first request that they be given a cathartic under such circumstances, even on the first night. However, they are assured that that will all be taken care of and that first of all a careful examination of the condition will be necessary in order to give proper medication. These patients are much concerned for the first few days, of course, the cathartic is not forthcoming, and much to their surprise, they begin to have normal evacuations of the bowels, perhaps not every day at first, but very soon, and eventually they inform you voluntarily that the condition is much improved. That is the rule in the majority of cases where there is no organic cause for the constipation. Only recently I had an experience with a patient whom I had previously treated, and who had had trouble of that sort, but apparently was not cured of the hysteria, and consequently was not cured of the constipation. The case was one of a chronic type of years standing, and beyond the age where we usually get good results in such cases. I received a telegram from this patient from some point in Florida, stating that the condition was very serious with complete obstruction of the small bowel, and that she had taken croton oil without results. I wired her and told her what to do, ordering absolute rest in bed and discontinuance of all medication. I have just received an answer that she is much improved. Apparently in hysteria the condition is caused by the same circumstances as the other symptoms of which those patients complain.

I wonder if there is not in all of these cases some definite psychic cause and while Dr. Marshall has given us the results which he obtained years ago, with apparently no recurrence, it is probable that the psychic element is eliminated. Where the condition is not a neurosis, and the patient is a normal individual, the condition may be due to bad mental hygiene. In cases of hysteria, where the neurosis is not cured and there is a return of the other symptoms there is a return of the constipation, and finding the cause of the neurosis will eliminate all of the symptoms, and consequently, the constipation.

Dr. Harold DeB. Barss: There are one or two points of interest to me. In my experience in the Surgical Clinic as I have examined a number of cases who come for indefinite stomach or bowel trouble, upon questioning them I find that the ideas of constipation among laymen vary. A large number definitely state that they are constipated, and yet there is no evidence to bear out their statements. Just the other day I asked a patient if he was constipated and he said, "yes." I said, "How long have

you gone without a bowel movement? Have you ever skipped a day?" He answered, "Yes, I did once, and I got a headache." This man took a cathartic every night. Patients of such type should be very amenable to treatment. I think another point of importance which was brought out is that we must first convince ourselves that there is no organic basis for the trouble. If we have eliminated every possible chance for an organic basis for the trouble, I think psychotherapy is a very good curative agent. I have to my credit one successful cure of constipation by the method outlined by Dr. Marshall. This patient who formerly used cathartics in considerable quantities, has not used any within the last two or three years.

Dr. James G. Van Zwaluwenburg: I have for a long time been convinced that the X-ray findings and the patients' statements of the condition often absolutely conflict. I doubt whether anyone knows whether he is constipated or not. I have reached a point where I don't know what constipation is. There must be some intoxication which arises from such a condition because I have experienced it. However, I refuse to state the habits of my bowels. I am not like a classmate of mine who claimed that his bowels moved regularly—every Friday morning. I believe that constipation is not of large bowel origin. However, I cannot with decency go any further since Dr. Marshall distinctly refuses to discuss the question of the entity of constipation. I merely want to express my appreciation of the presentation of the subject tonight.

Dr. Marshall (closing the discussion): Dr. Klingmann's point is well taken in regard to constipation occurring as a feature of hysteria or some other neurosis. Of course in such a case, the neurosis is the thing which should be treated. I haven't any doubt but that some of the cases I have treated fall into that group, but it is not always convenient to treat the underlying condition scientifically any more than it is always feasible to perform a surgical operation for the underlying cause of constipation. The treatment after all is only a symptomatic form of treatment and is comparable to the symtomatic treatment of hysteria by the electric current rather than treating the patient by psychoanalysis, which eliminates the cause of the trouble. In all the cases with which I have come in contact, a large majority of them fall into the group which Dr. Barss mentioned, those patients who are not constipated at all, but only imagine they are.

The subject has been a very interesting one. I must admit that I have not treated a great many cases. In the cases in which I have taken up the treatment seriously, I have had no failures except the one which I have reported, in which there was a partial result. Many times I have used the method in cases that were being treated for other conditions in which constipation was incidental. In such cases there has been improvement but not a complete cure, because the treatment was not undertaken with proper care.

# REPORT OF TWO CASES OF THYROADENOMA OF THE POSTERIOR PHARYNX AND NOSE.

A. C. Furstenberg, M.D.

(From the Department of Otolaryngology, University Hospital, Ann Arbor, Michigan).

Thyroid metastasis to the upper respiratory tract is a very rare occurrence but not infrequently remarkable glandular tissues growing within the bones of the skeleton, resembling structurally young or growing thyroid gland have been encountered. From a pathologic viewpoint these tumors are thyroadenomas and structually of a benign type, but clinically they are found to grow rapidly, eroding the bone and tissues in which they occur and manifesting malignant properties. It is, moreover, an interesting fact that metastasis has frequently occurred when a careful study of the thyroid gland itself has failed to show any evidence of a primary tumor. The metastatic focus may not only resemble the structure of normal thyroid but may also be capable of assuming the same function. In this connection Von Eiselsberg reported an interesting case in which symptoms of cachexia appeared following the removal of a goiter but prompt improvement occurred with the appearance of a tumor in the sternum two years later. Because of symptoms of pressure it was necessary to remove the tumor and sternum four years later, following which, the patient developed cachexia and death finally occurred from marasmus.

As to the cause of the development of metastasis, according to Adami because of the distance of the bones affected from the thyroid it does not seem reasonable to advance the theory of fetal inclusion, that is, to suppose that we are dealing with cases in which, during fetal life, pieces of thyroid became detached and lodged in growing bone to lie dormant for years and suddenly take on an active growth. He has pointed out that there can be detected in the thyroid even during adult life small accumulations of cells called "mother cells" by Wolfler capable of growing and producing new acini. He believes, therefore, that the simplist explanation for metastasis is that certain of these cells gain entrance to the circulation and are carried through the organism to lodge in tissue favorable to their growth.

The case reports are as follows:

Mrs. H., age 45, entered the Hospital complaining of difficult nasal respiration. Her family and personal history were of no importance. Her present trouble began two years ago when she gradually became hoarse, suffered from pain in the right eye, and a few weeks later was seized with frequent attacks of epistaxis. She consulted her physician who removed a growth from the right nares following which nasal breathing became free but intermittant hemorrhages continued. Three months later it was necessary to submit to a second operation, the growth having returned and epistaxis more frequent. She remained under the constant care of a nose specialist for three months during which time she was free from symptoms. The nasal obstruction and epistaxis, however, returned, the hemorrhages became alarming, she suffered extreme weakness from the loss of blood and was seized with repeated attacks of acute suppurative otitis media.

On entrance to the Hospital the following physical signs of importance were as follows: Patient had an exophthalmos more marked on the right than on the left. There was an enlargement of the right lobe of the thyroid. There was a distinct tremor of fine character in both hands. The heart action was rapid with a systolic murmur heard at the aortic area. Blood pressure 175. Hemoglobin 55 per cent. The eye examination was negative except for a slight edema of both discs and a proptosis of 21 in OD and 18 in OS. The patient was a typical mouth breather. The right side of the nose was found completely filled with a soft red spongy like tumor extending back into the nasopharynx. The left side was free from the tumor mass. A section of the neoplasm removed for pathologic examination was reported as follows: Tissue presents character of thyroid adenoma without colloid Large hypertrophic cells line the follicles like those of exophthalmic thyroid. May be misplaced thyroid tissue or thyroid adenoma metastasis. Should be treated as a carcinoma.

Following this report the tumor mass was radically removed under cocaine and adrenalin and the nasal cavity firmly packed with gauze. The following day the packing was removed and during the following ten days that the patient remained in the Hospital, she enjoyed unobstructed nasal respiration and was free from hemorrhage.

The second case, Mrs. P., age 40, entered the Hospital complaining of difficult breathing and hoarseness. She was assigned to the Medical Out Patient Department where the following history was obtained.

Family and personal history of no importance. Present illness began in 1903 following childbirth at which time she noticed a hard tumor mass in the neck associated with a dry hacking cough. Since this time she has never been entirely free from the cough and the swelling in the neck has been slowly but progressingly increasing in size. About six weeks ago she was seized with a severe attack of sore throat associated with chills and fever, difficult respiration, painful and altered phonation and the expectoration of large quantities of mucopurulent secretion mixed with portions of membrane and streaked with bright red blood. Htr condition was diagnosed as diphtheria by her home physician and antitoxin administered. Since this attack she has noticed a progressingly increasing dyspnea and marked alteration of voice. Physical examination in the Medical Clinic revealed a large bilateral goiter. The heart and lungs were negative. Blood pressure 170, systolic, 110 diastolic. Urine showed a trace of albumen. Wassermann negative. She was referred to the Department of Otolaryngology where examination of the throat revealed a large tumor mass of the posterior pharyngeal wall, the anterior convexity extending forward almost to the tip of the epiglottis. The tumor was covered by normal mucous membrane and showed no evidences of ulceration. On palpation it was hard and firm and presented no characteristics of fluctuation. The larynx showed congestion and infiltration of the vocal cords characteristic of a chronic laryngitis. There was no paralysis of the intrinsic laryngeal muscles. The X-ray plate showed an enormous swelling of the retropharyngeal tissues encroaching upon both the nasal and aural pharynx and extending from the level of the second cervical vertebra above downward to the level of the fourth cervical.

Because of the onset history of a severe pharyngitis immediately followed by the difficulty in respiration it was reasonable to assume that we were dealing with a retropharyngeal abscess even though the firm hard character of the mass on palpation failed to substantiate that diagnosis. The posterior pharyngeal wall was aspirated with the result that only a few drops of blood was obtained. The mass was incised at the inferior border when it was found that the knife had penetrated a large new growth. Hemorrhage was profuse. A small piece of tissue removed for pathologic examination was reported as thyroadenoma.

As to the treatment of thyroadenoma, it is the consensus of opinion among surgeons that the metastatic focus should be treated surgically by radical extirpation. Because of its clinical manifestations it is malignant and should be treated as one would treat a carcinoma. In the first case reported, radical removal of the tumor from the nose was possible. In the second case in which the tumor involved the posterior pharyngeal wall, its enormous size, its location and vascularity, made it inaccessable either by external operation, or by surgical interference through the mouth.

Because of the short time in which the patient has been under observation the report of the last case with reference to treatment is in-

complete. We hope to report the therapeutic measures used and results in a later communication to the Society.

DISCUSSION.

Dr. James G. Van Zwaluwenburg: Dr. Furstenber, whether intentionally or inadvertently, failed to read what to me is the most interesting part of my report to him. That which is not so obvious about the plate is the fact that the atlas is very much reduced in heighth; that the head is extended on the neck so that the spinous process of the axis is very much nearer the occiput than ordinarily. Also I thought I could see some irregularities in the body of the axis. You will note this sharp projection, and also the wearing off of the corner below. My idea of this, taken in conjunction with the enormous mass of the tumor, is that this is a Pott's, and the enlargement was secondary to it. I think that if Dr. Furstenberg could look a little more deeply he would probably find that there is an involvement of the bony tissue as well as of the soft tissue of the neck. Personally I cannot see how the soft tissue alone could produce all of this disturbance. There must be destruction of the lateral body of the axis.

Dr. Furstenburg (closing the discussion): From the fact that Dr. Van Zwaluwenburg has shown us a partialogic condition in the atlas, it may be assumed that the thyroid metastasis occurred first in the bone, because it is very rare that thyroid metastasis has occurred in the soft tissues of the body, but it is quite frequently found in the bones, especially the bones of the head, sternum, clavicle, and scapula. For this reason it may be possible that the metastasis occurred to the atlats, and then by extending downward, presented itself in the pharynx.

# TRANSACTIONS

OF THE

# Clinical Society of the University of Michigan

## Stated Meeting, February 6, 1918

**The President, JAMES G. VAN ZWALUWENBURG, M.D., in the Chair**
**Reported by REUBEN PETERSON, M.D., Secretary**

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## REPORT OF A CASE OF CICATRICIAL ECTROPION CORRECTED BY PLASTIC OPERATION.

GEORGE SLOCUM, M.D.

(From the Ophthalmic Clinic, University Hospital, Ann Arbor, Michigan).

H. H., aged 35, entered the Clinic of Ophthalmic Surgery of the University Hospital December 4, 1917. The lower eyelid and face below the outer angle of the eye were burned when ten years of age,

*Examination.*—Marked cicatricial contraction of the lower lid O. D., the deformity so great that the eye lashes touched the cheek, the outer half of the lid showing the greater deformity. There was a marked hypertrophic conjunctivitis, the exposed mucous surface presenting a marked convexity forward, a condition which nearly always follows prolonged exposure of the palpebral conjunctiva.

December 6, 1917. Plastic operation. Incision along the lower lid border about 2mm. from the

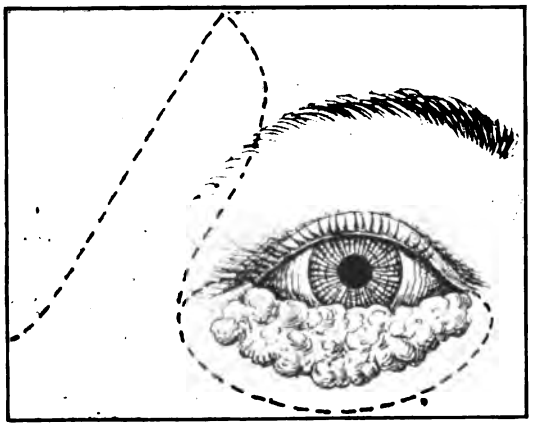

**Fig. 1. Position of flap in this case. Blasius operation. (After Beard).**

eye lashes extending to just below the puncta lacrymalis, dissection carried up toward the orbital margin, freeing the lid to septum orbital until it could be restored to its normal position. Blasius flap removed from the temple turned down and sutured in position filling the gap caused by the restoration of the lid to a normal position, no tension on the flap being permitted. The denuded area was covered by undermining the adjacent skin and suturing.

Figure 1 represents the position of the flap taken

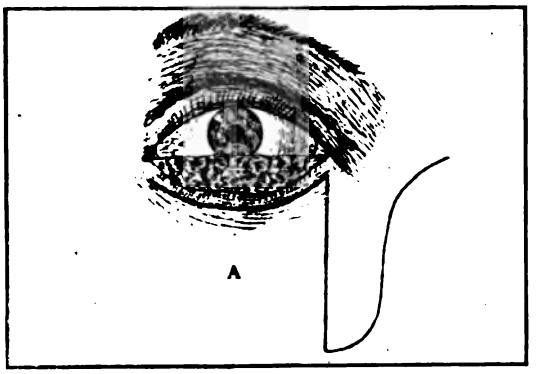


**Fig. 2. Von Lagenbeck's operation for ectropion. A. Incision. Ball's Ophthalmology.**

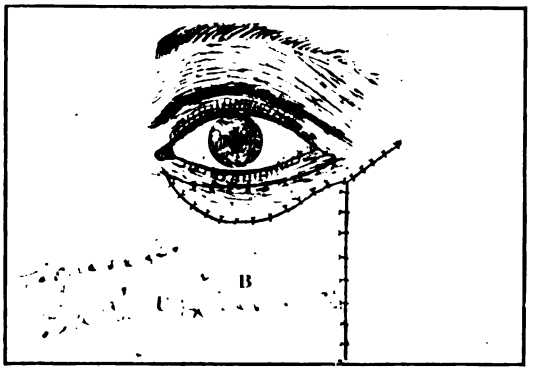


**Fig. 3. Von Langenbeck's operation for ectropion B. After suturing.**

as in the Blasius operation from the temple near the eyebrow. Figure 2 represents the incision along the eyelid border after Von Lagenbeck, freeing the lid border from the cheek so that it could be re-

stored to its normal position. Figure 3 represents the appearance of the flap in position in the Von Lagenbeck operation in which the flap is taken from the cheek. The operation performed in this case is a combination of the Blasius and Von Lagenbeck operations. From this operation a partial restoration of the lid was obtained, but owing to too great laxity of the lid border and the hypertrophic conjunctivitis, the deformity was changed from a

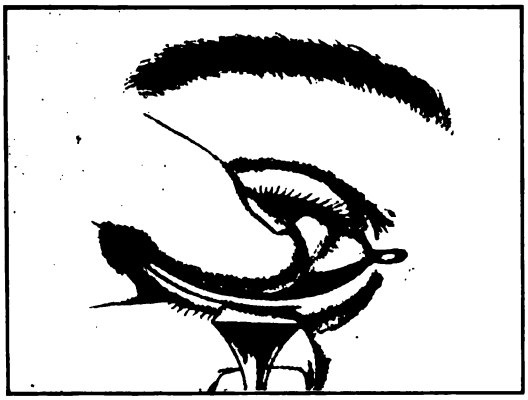

FIGURE 4.

cicatricial ectropion with marked stretching and displacement of the whole lower lid to a flaccid ectropion with hypertrophic conjunctivitis.

After the flap had entirely healed the hypertrophic conjunctiva was reduced by treatment with alum pencil. When palliative methods fail in hypertrophic conjunctivitis the hypertrophy may be reduced under cocaine anesthesia by the Ziegler cautery operation which consists in making a series

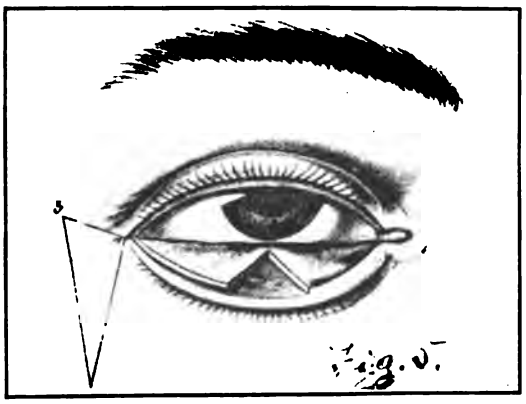

FIGURE 5.

of cautery punctures in the hypertrophic conjunctiva paralleling the lid border about 3 mm. from the sharp inner margin.

*Second Operation.*—In this case the remaining flaccid ectropion with the hypertrophic conjunctivitis one-half to two-thirds reduced, was corrected by means of the Kuhnt-Szymanowski operation. The technic of this operation may be outlined as follows: The eyelid is split along the intermarginal line from near the center of the lid to the outer canthus. Next, the skin is divided from the outer canthus outward and well upward ten to fifteen millimeters; from the outer extremity of this incision a second incision is continued downward about fifteen mil-

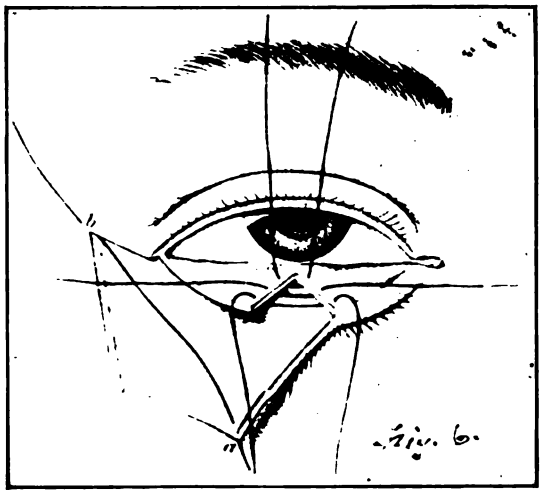

FIGURE 6.

limeters, the trend of the incision being mesialward until nearly below the outer canthus where it turns at an acute angle up to the outer canthus. The wedge shaped piece of skin thus outlined is removed; the skin of the lower lid is now dissected downward until the outer angle of the undermined skin can be carried outward and upward far enough to cover the denuded area. A wedge shaped piece of mucous membrane is next removed from the mesial portion of the conjunctivo-tarsal flap. Sutures are introduced in the borders of this wedge shaped area so that they may be tied on the conjunctival surface. The skin flap is next sutured in place filling the

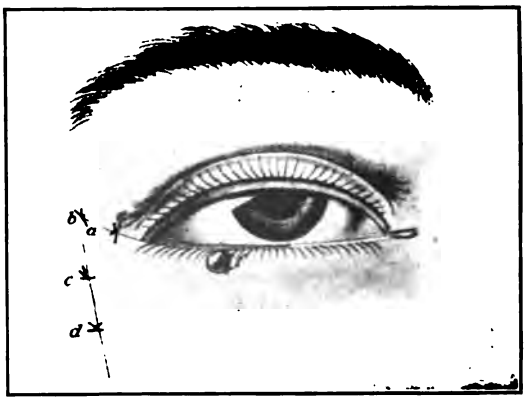

Fig. 7. Kuhnt-Szymanowski operation. Appearance after the operation. The lower lid lies in its proper position; 4 (ab., c., d., e.) sutures suffice for the fixation of the flap. The suture for the fixation of the skin to the tarsus is tied over a bead (f).

denuded area beyond the outer canthus: a through and through suture holds the conjunctival and skin flaps in coaptation and prevents separation by clots

or exudate. Border sutures may unite the skin and mucous borders of the lids when it seems advisable, provided they are not allowed to remain longer than forty-eight hours. If they remain much longer they are liable to cut and produce fissuring of the lid border. Figures 4, 5, 6 and 7 taken from Meller's Ophthalmic Surgery, show the steps of the operation.

The photographs below show the condition before each operation and the final result.

Fig. 8. Before first operation and after second operation.

DISCUSSION.

Dr. Robert H. Baker: I would like to ask Dr. Slocum what suture material was used on the conjunctival surface.

Dr. Slocum: No. 1 silk.

Dr. Baker: How do you control the irritation against the eyeball?

Dr. Slocum: There is very little irritation if small sutures are used.

Dr. Peet: Do you use vaseline on your sutures?

Dr. Slocum: No. I can see no objection to its use, however; the mucous secretion covers the sutures very quickly and the patient is not usually annoyed by their presence.

I might explain why we did not use a skin graft. The Tiersch graft is so thin that it gives but little support. They are, therefore, most successful when support is not needed, particularly in the upper lid. In this case we not only had a deformity to correct, but in addition a tendency of the lid to drop down from the greatly increased weight; the orbicularis muscle was to a large degree atrophied and there was marked loss of skin area due to cicatricial contraction; it was, therefore, essential that the lid should be supported from below. Because of the increased length of the lid and the stretching of the outer canthus, the second operation was necessary in order to hold the lid against the eyeball. In ordinary ectropion, due to laxity of the tissues and rolling outward of the lid without shortening of the skin below, the second operation would be sufficient.

Dr. Max Peet: This is certainly a beautiful result of a plastic operation. I firmly believe in the use of pedunculated flaps in preference to skin grafts where they can be used. Skin grafts have a habit of not growing sometimes, and with pedunculated flaps you can have thick flaps. The only way you can get this in a skin graft is to use the Wolfe grafts and they are poor to take. For ordinary skin grafting the Tiersch or Reverdin grafts are about all we have outside of a pedunculated flap such as was used here. And certainly for a condition of this type, using either Tiersch or Reverdin grafts could not give as good a result as has been obtained. The Reverdin graft especially would be useless because you would simply have a mass of small points of skin, some of them growing, and some not, with spotting, the old skin white and the new skin bluish. Some do not like the Reverdin graft and regard it as useless. Personally I think we have cases where it is very useful. However, in eye work it seems to me that pedunculated grafts such as this would give the very best results. I could not ask for a better result than Dr. Slocum has obtained in this case.

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## REPORT OF A CASE OF AN ENORMOUS POSTOPERATIVE VENTRAL HERNIA.

Leslie L. Bottsford, M.D.
(From the Obstetric and Gynecologic Clinic, University Hospital, Ann Arbor, Michigan).

Small or moderate sized umbilical or ventral hernias are of common occurrence and are usually easily repaired, although at times a large area of skin and fat is necessarily removed.

Enormous ventral hernias, as well as the inguinal, scrotal, femoral and labial varieties, are relatively uncommon and present an entirely different operative problem. Even if operable, they are associated with a very high mortality and morbidity. In the cases of large or massive and below the enormous ones, the estimated death rate has been placed at 6 per cent. (McGannan). However, when strangulation or obstruction, is added, the mortality in massive hernias reaches nearly 50 per cent. Correspondingly we should expect even a higher rate for the enormous type, one of which I wish to present.

In the usual simple hernias postoperative treatment extends over about 18-21 days; in massive hernia, 50 days; and after enormous hernias, from 50-150 days. The causes of

mortality in the enormous hernias have been classified by Hewitt into several groups.

I. Those due to cardiovascular-renal conditions, acute dilatation of the heart, apoplexy, uremia, pulmonary edema, embolism and thrombosis.

II. Postoperative abdominal conditions: obstruction, acute dilatation of the stomach, peritonitis, and suppuration of the abdominal wall.

III. Those due to strangulation: toxemia of obstruction, peritonitis from ruptured and gangrenous bowel and embolism of the mesenteric arteries.

IV. Shock due to loss of blood, anesthetic, exposure or manipulation of the hernial contents.

Of these various dangers, probably the most usual one encountered is acute cardiac failure. In enormous hernias, since the patients carry a considerable portion of the abdominal viscera outside the normal habitat, there is a consequential loss of pressure on the large splanchnic vessels, resulting in their dilatation and associated with compensatory changes in the general circulation, which sustains the blood pressure. In addition, most of these patients have some cardiovascular-renal disease, which also further embarasses the heart.

When an attempt is made to return the protruded viscera to the abdomen, followed by repair of the hernial opening, a readjustment of the vascular conditions in the abdomen must occur. Before the latter can be effected, the heart must overcome a great increase in its ordinary burden, and if any myocardial degeneration is present, rapid dilatation usually follows.

Again, in returning the viscera to the abdomen, the intraabdominal pressure is necessarily enormously increased, following which jaundice or gastrointestinal hemorrhage, with or without perforation, may ensue, even though the heart temporarily upholds the added burden and withstands the marked engorgement of itself and the lungs.

To these dangers must be added all those previously mentioned, and as a result, it can be readily understood that the operative mortality must be high, even in selected groups of cases.

Barker, in speaking of voluminous hernias, advises rest in bed, limited or restricted diet, and daily attempts to reduce the mass partly or wholly. When these methods fail, he rejects the patient, as unfit for operation. He also strongly recommends local and spinal anesthesia, in view of the consecutive lung, liver, and kidney troubles.

Rest in bed, blood pressure, pulse readings and phthalein estimations with careful urinary observation for twenty-four hour periods prior to operation are of value. A snugly fitting abdominal binder, which is gradually tightened, while the blood pressure, pulse, and respiration are observed, is sometimes of aid. Preliminary treatment along these lines may be carried on for several weeks preceding operation.

I shall not discuss the various types of operations which have been devised for the cure of these hernias, more than to mention them. The type of operation most suitable must needs be adopted for each individual case. They have been treated by extensive flap formation with layer to layer or overlapping suturing: either alone or combined with no less extensive muscle and flap sliding; or by autoplastic fascial transplants; or by pedunculated fascial flaps; or by the re-enforcement of such areas by the use of the various wire filigree screens.

In addition to these, we have Haynes' inversion of the hernial sac by interrupted sutures with coaptation and union of the abdominal wall adjacent to the hernial orifice. The sac may or may not be opened for the placing of the first sutures, as seems desirable.

Hewitt in a recent article advises colectomy with subsequent end-to-lateral or end-to-end anastomosis of the ileum and sigmoid, removing in this way a large part of the mass to be returned to the abdomen. Portions of the omentum may be also removed, but the removal of a large mass of omentum may so damage by embolism the circulation of the stomach walls, that ulceration and hemorrhage will follow.

These patients with enormous ventral hernia are usually obese, averaging from 200-300 pounds. The hernias are usually postoperative below the umbilicus, and have existed for some years, operation usually being sought as a last resort to secure relief from intestinal disturbances and the drag, pain, or discomfort of the mass. The sac may be single or multilocular and the hernial orifice varies greatly in size the largest on record as repaired being eight by five inches in its longest and broadest diameters.

In a hasty review of the recent literature, I have been unable to find a case as obese or with as large and complicated a hernial mass as the one I wish to present.

### CASE REPORT.

This patient is 49 years of age and presented herself for examination Oct. 26, 1917. Her parents and near relatives have all been very obese, weighing from 225-315 pounds. When seen in the Clinic the patient weighed 356 pounds, having gradually increased in weight from 160 pounds since 1900. Her history is entirely unessential except from an operative standpoint and this is based upon her personal statements. In 1904 she underwent a

laparotomy at which time a fibroid tumor of unknown attachment and weighing 39 pounds was removed through a transverse incision below the umbilicus. She was reoperated in 1907 and a second fibroid weighing 40 pounds removed. In 1909 she was delivered by abdominal Cesarean section of a 7½ pound infant and immediately following the Cesarean, a tumor weighing 42 pounds was removed Both of the latter incisions were above the umbilicus. Three months following the last operation, the patient begun to develop a hernial mass in the midline slightly below the umbilicus, which has gradually increased in size up until the present, now occupying practically the entire abdomen and forming a pendulous mass, which falls nearly to knee level when she is on her feet. The large, distended loops of intestine are clearly seen in the photograph lying just beneath the skin. During these years she has had no trouble except for some tympanites at times following errors in diet, and the inconvenience from the size and location of the mass itself. The bowel movements have been perfectly regular and her appetite voracious. When asked why she sought surgical advice, she volunteered the information that she was tired of carrying the mass around, inasmuch as she had to perform all her own household duties.

Needless to say this is a truly inoperable case from several standpoints: First, from the size of the hernia, for it was impossible to palpate any limits to the hernial orifice; second, the intestines were all greatly dilated and apparently densely adherent to the sac; third, the patient was of the cardiovascular-renal type with a serious hypertension. All three of the factors definitely contraindicated any operative interference. Some years ago surgical aid might perhaps have been rendered.

### REFERENCES.


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2. Hewitt, W. R. "Enormous Hernia." Surgery, Gynecology and Obstetrics, Chicago, 1917, XXIX, 215-221.


### DISCUSSION.

Dr. Cyrenus G. Darling: In discussing this paper I want to speak of some of the objections which have been raised, not particularly to this case but in relation to ventral hernia in general. One must not say that any case of veneral hernia is inoperable until he has carefully studied the patient and learned something of the size of the opening, the amount of tissue that has been destroyed, or rendered useless for approximation in the particular case, or knows something about the cardiovascular system and is sure that the cardiovascular condition does not depend upon the method of life of the patient rather than upon a particular disease.

The death rate in ventral hernia has probably been exaggerated because of the lack of preparation before the operation is done. The success of such an operation depends largely upon these two things.

There is one point upon which the doctor touched slightly and that is the choice of an anesthetic. He mentioned the use of spinal anesthesia. Now, the use of ether as an anesthetic may not be proper in these cases and in cases where I had any fear of ether being a contraindication, I have used gas anesthesia. In one case where two operations had been done and both of these torn open by the patient vomiting for a week after an ether anesthetic, I used gas without any disturbance whatever and with a perfect result as far as repair of the hernia was concerned, and I have used gas in several other cases since to avoid the accident of vomiting, or in case there should be any renal disease.

Another complication which may lead to a fatal result is edema of the lungs or pneumonia, which Mayo pointed out years ago in operations for umbilical hernia, as being due to the restricted respiration brought about by bringing the parts together.

This is true in those cases which are repaired by bringing the lateral sides together in the median line rather than transversely. So we find that if we are to be successful in the repair of ventral hernia we must avoid this longitudinal repair as much as possible, depending upon a partial transverse closure even if the general opening is longitudinal.

There is no reason why a person with a great amount of fat cannot lose a great deal of it by rest in bed and diet. This may also have the effect of pressure upon the vessels within corrected to a certain extent if the tumor or pressure is reduced and a properly adjusted binder is applied. The patient in the course of four or five weeks will become accustomed to this pressure. The trouble is that very few of these persons find their hernias sufficiently bad to submit to this treatment. A ventral hernia which has existed for a long time has probably led to atrophy of the abdominal structures and the loss of abdominal compression upon the abdominal contents has allowed the fat to increase and distention to increase, and we must carefully get rid of this distention if we are going to bring about a good result.

The next thing to consider in the operation will be the method of securing sufficient tissue to bring about a good repair. Mention was made of the use of the hernial sac as part of this tissue. Now the hernial sac may be used for this purpose, but in no instance should a hernial sac be so used unless the hernia has been opened and it has been ascertained that there are no adhesions to the sac or to the ring. After you have found all the underlying tissue separated from the sac, there is no reason why you should not employ this for the purpose of building up a wall. The method of forming a transverse approximation should next be considered, as very few of these incisions are made transversely. Nearly all of them are longitudinal, and mostly below the umbilicus. The hernia as it is formed may be a round opening. In approximating this you can usually, by putting stitches in, from above downward, and tying them, bring the lower part nearly to the upper, converting this almost into a transverse incision. The object is to allow more space anteroposteriorly than is possible if you approximate from the other direction. In doing that you will probably bend upon themselves the recti muscles, but while you are shortening them you are bringing them in towards the median line,, and I think the success depends upon getting the approach to the transverse coaptation.

The next important point is in maintaining this position. We have used kangaroo tendon, chromacized gut and silver wire. In most of the cases where I have used silver wire I have obtained very good results. Some of the sutures are buried and some are put in on the outside as stay sutures and are allowed to stay for two or three weeks. These act as a support to the wall. They may be placed back through the skin and through the tissues and brought out some distance below. We usually have two of these some distance back on either side and they are then tied around a piece of gauze. These act as regular splints for the support of the tissues during the time of repair and it matters not which way you apply them, according as the conditions demand. I have found that by using these supports during the period of formation of tissue or repair, I have been able to maintain it much better than by any other method. Even in cases of suppuration where I have been obliged to work upon infected tissue, I have succeeded in getting results with these supports. So I look upon these silver wire sutures as quite essential in the process of repair.

I think that any type of operation is applicable. I see no reason why, if tissue is wanted, we may not import a fascial flap though I believe that transplanting fascia here is liable to lead you astray in not using tissue at hand. In making a layer dissection you can treat the layers between the superficial fascia as one layer, or you can use them separately. You make your approximation usually with the deep fascia and bring the muscular layer over perhaps using two layers to get a good approximation.

Ventral hernia should be repaired as early as possible after its discovery before it reaches the enormous size shown here. It should never be permitted to go so far before repair has been undertaken unless there is some unusual reason for leaving it. The death rate associated with ventral hernia as given in the text books is misleading. You are told of various accidents which apply to any abdominal operation and they are put up against ventral hernia. You might say that if you had carefully selected cases you could lower this death rate to one per cent. If you were to take a hundred cases such as the one exhibited here and attempt to operate upon them your death rate might be a trifle over 6 per cent. Ventral hernia is no more a dangerous operation so far as the operation itself is concerned, than other forms of hernia of equal size, and if you had all of these other complications which have been mentioned as the cause of the high death rate, you would not care about operating those cases even with ordinary inguinal hernia. If you operate recklessly all cases of hernia as they come along, you will have a 6 per cent. death rate, no doubt. It would be misleading to say that that should occur of itself if you kept in mind the danger of the anesthetic, the method of approximation, the preparation of the patient prior to operation, the care in approximating the tissues without including in your sutures any of the gut or omentum, and if you don't leave spaces between your sutures where a piece of gut may get in and form an obstruction. All depends upon the care with which you do the operation.

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## SYPHILITIC PARALYSIS OF THE FIFTH CRANIAL NERVE.

CARL D. CAMP, M.D.

(From the Neurologic Clinic, University Hospital, Ann Arbor, Michigan).

Isolated paralysis of one of the cranial nerves as a manifestation of syphilitic infection is not infrequent in the cases of the third and sixth nerves and in fact, in the case of the former, is so frequent as to justify the statement of Fournier that "paralysis of the third nerve is

the sign manual of syphilis." There are a few other causes of third nerve palsy. In the case of the sixth nerve the number of cases is even greater but owing to the fact that there are so many other causes of sixth nerve palsy it is not diagnostic of syphilis. The fourth cranial nerve can also be paralysed in syphilis but the recognition of the condition is not so easy as in the case of the first two mentioned.

The other cranial nerves seem to be much less frequently affected although a slight disturbance in the function of the eighth, a shortening of bone conduction, is common. Facial, or seventh nerve palsy, is much more frequently due to syphilis than is supposed and I have seen six cases of it in the past two years. It usually occurs in the early stages of the infection and is transient.

Fifth nerve palsy due to syphilis seems not to be well known, though there are a number of references to its occurrence in the literature. As in the case of the other cranial nerves there seems to be two distinct types of involvement; the first occurs soon after an infection and is usually isolated and transient, yielding readily to treatment; the second occurs late in the disease, is usually accompanied by other paralysis and is resistant to treatment. This second type is the kind reported as occurring in association with tabes. In 1905 I called attention to the implication of the fifth nerve in cases of tabes examined pathologically (*Univ. of Penna., Med. Bull.*, Jan., 1905) and said "The involvement of the fifth nerve is not very uncommon in tabes. Spiller has seen a degeneration of the fifth nerves in at least four or five cases studied pathologically. Most authors refer to the painless dropping out of the teeth in this connection. According to v. Leyden and Goldscheider the first cases were reported by L'Abbe (1868) and by Dolbean (1869). The falling out of the teeth is followed by the atrophy of the alveolar processes of both the upper and the lower jaws. Necroses of the jaw may occur and sequestra form. The process is at first painless, but infection usually occurs later. Sensory disturbances in the distribution of the fifth nerve may or may not be present. The most frequent pathologic finding is a degeneration of the descending spinal root of the fifth nerve." At the same time I reported a case of tabes in which one of the symptoms was recurring attacks of herpes zoster in the distribution of the fifth nerve and this was followed by painless falling out of the teeth.

In 1910, Dr. William G. Spiller and myself reported a case of syphilitic paralysis of the trigeminal nerve (*Amer. Jour. of the Medical Sciences,* March, 1910) which had been studied clinically by Dr. Spiller five years before and which we together studied pathologically. Section of the roots of the right fifth nerve showed much degeneration when stained by the Weigert method. In this case there was also a right hemiplegia due to recent softening in the temporal lobe. Similar cases have been recorded by Oppenheim, Brasch and by Pick. The last two also found the nerve degenerated and a degeneration of the spinal nerve root. The trouble is usually unilateral, but bilateral involvement has been reported by Leudet and Labarriere and by Hutchinson The sensory division is much more frequently affected than the motor though cases with distinct palsy of the muscles of mastication have been reported by Ziemsson, Lowenfield and Oppenheim.

It will be noticed that the cases cited above occurred in the later stages of the disease and that the palsy was more or less permanent. As in the case of facial palsy, however, the palsy can occur in the early stage and is then transient but these cases are rare, much more so than the facial and, when they occur, usually affect the sensory division and give rise to neuralgic pains so that these cases are, in effect, cases of trifacial neuralgia. The pathologic change is probably a basal meningitis of moderate severity as is the case in the early facial palsies.

The case that I wish to report is one of fifth nerve palsy occurring in the early stage of syphilis which began with pain in the face; later he developed the anesthesia and palsy characteristic of the degenerative change in the nerve. He improved rapidly under antisyphilitic treatment.

Mr. L. H., age 39 years, was admitted to the University Hospital Jan. 21, 1918, complaining that the right side of his face felt "dead." His family and previous medical history were not important in this connection except that he said that he had been infected with syphilis about four months before. About five weeks before he was admitted to the Hospital he began having a pain in the right cheek which at first was thought to be due to a tooth, but soon afterward the pain spread to the right side of the head and to the nose and eye. An X-ray, he says, showed no trouble in the teeth and as the pain was severe a physician treated his antrum, but was unable to find any pathologic condition there.

He says that shortly after this treatment he developed a numb feeling in the entire right side of the face and the right side of the head and it was that way up to the time of his admission although

the pain had subsided. He had no other complaints.

Examination showed that he was well nourished and he seemed to be mentally normal. There were some enlarged glands in the postcervical region. His pupils were equal but they did not react to light. They reacted well in accommodation. The extraocular movements were normal. There was no facial palsy, he could wrinkle the forehead, close the eyes and draw back both corners of the mouth equally well. He was anesthetic to touch and pinprick in an area corresponding to the distribution of the right fifth nerve. The jaw on opening deviated to the right, but the tongue protruded in the midline of the jaw. The right masseter and temporal muscles could not be felt to contract in the act of biting, though the muscles on the other side contracted strongly. The right cornea was anesthetic. There was no deafness, and no difficulty in speech or deglutition. His tendon reflexes were all normal and there was no sensory loss over the body or extremities.

The ophthalmoscopic examination showed a marked neuroretinitis in the right eye of a rather acute type, the left fundus was somewhat congested, but otherwise normal.

The cerebrospinal fluid showed 79 cells to the cmm. The carbolic test was negative. The Nonne-Apelt phase one was positive, phase two negative. Reducing substance was normal. The gold sol and the mastic reaction gave the curve for cerebrospinal syphilis (122321000). The Wassermann reaction on the cerebrospinal fluid was 4 plus. The Wassermann reaction on the blood was also 4 plus.

The patient was placed on treatment January 22 consisting of daily intramuscular injections of mercury succinumide. On January 20 he said that his face was not so numb and the area of anesthesia was found to be diminished in size. On February 4 he was completely reexamined and the area of anesthesia was found to be still further diminished. The distribution of the third division and the auriculotemporal branch showed no sensory change. The evidence of motor palsy was the same as before.

The patient continued to take treatment until February 9. At this time the anesthesia of the face had almost completely disappeared and the right masseter muscle would contract, although it was weaker than the left.

DISCUSSION.

Dr. Max Peet: This is especially interesting to me naturally because the man first had the symptoms of trifacial neuralgia followed by anesthesia. Some of these cases certainly have pain in the face for many months or many years due to syphilis and come in for operative relief. All of our patients so far that we have operated upon have had negative Wassermanns fortunately, but the possibility must always be considered. It seems to me extremely interesting that we have acute inflammation of the fifth nerve giving the acute pain of trifacial neuralgia followed so rapidly by degenerative changes. I was impressed with the drawing showing such a complete distribution of anesthesia, more than in any of the cases in which we have cut the sensory root. All of those cases have shown diminished sensation over the pinna of the ear but not complete anesthesia, so the man has really a greater anesthesia than we often get by cutting the sensory root.

Dr. Camp: With reference to the sensory distribution of the fifth, it is, of course, a rather variable factor. In cases where the gasserian ganglion has been extirpated, sometimes the ear is completely anesthetic including the pinna, sometimes the area of anesthesia comes through the ear, and sometimes the ear escapes entirely.

# TRANSACTIONS

OF THE

# Clinical Society of the University of Michigan

## Stated Meeting, March 6, 1918

**The President, JAMES G. VAN ZWALUWENBURG, M.D., in the Chair**
**Reported by REUBEN PETERSON, M.D., Secretary**

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## CONSIDERATION OF SOME DENTAL ANOMALIES.

CHALMERS J. LYONS, D.D.Sc.
(From the Dental Clinic, University Hospital, Ann Arbor, Michigan.)

More than a quarter of a century ago it was recognized that during the eruption of the teeth, the brain and the nervous system, the stomach and whole intestinal tract, the circulatory and respiratory systems, all show an increased, though varying liability to irritation.

Years ago that great master in Oral Surgery, Dr. James E. Garretson, taught that the process of the eruption of teeth caused many disorders through reflex disturbances in various directions.

For years unerupted teeth have been recognized as etiologic factors in the production of cysts of the jaws. In spite of all this knowledge little attention has been given to the subject of dental anomalies by writers upon surgical topics. The comparative scarcity of literature along this line has perhaps led to misapprehension regarding diagnosis and treatment and conservative methods, which would result in benefit to the patient have too often given place to uncalled for radical operations, obviously detrimental to the comfort and appearance of the patient.

It is to some of these abnormal conditions that the writer purposes to call your attention at this time.

The human family has, through centuries of use, established what we call a normal condition of the occlusion and arrangements of the dental organs. It has been so established because it is a condition that is most favorable for their function.

Now when a variation from the normal occurs which is a disadvantage or a disturbance of function, we naturally look back to discover, if possible, the cause of the deviation from normality. It has been contended, although not clinically proven, that there is a tendency to degeneration of the jaws of the human race and that the continual development of the nervous system is accomplished at the expense of the osseous system. Thus the more intellectual the human race becomes, through the process of evolution, just in that proportion will the osseous system become depleted.

The disciples of Darwin believe that as we ascend the scale in so-called civilization, we lose some of the factors that were necessary for the maintenance of life in the primitive man. As an illustration—the animal depends upon its physical strength for its food and its protection. The bony framework comparatively is very strong. The bones are large, long and the prominences upon their surfaces for the attachment of muscles are very marked, thus indicating that the muscles are large and strong. As we ascend the scale to man, we find that the bones are comparatively small in diameter, and shorter, showing that the muscles are not so large and strong.

Great changes take place in the jaws and teeth. They become smaller in size, the teeth change their shape, because man is now no longer required to procure his food with his teeth or to protect himself with his jaws. The temporal muscle, zygomatic arch and jaws decrease in size and are not so prominent. Talbot has stated that if we compare the jaws of the orang and chimpanzee with those of the

Esquimaux, we observe a wonderful difference in regard to size, shape and character of the jaws and teeth. If we now compare the jaws of the Esquimaux with those of our working classes, and these latter with the jaws of young people of the well to do classes who are not required to work physically, we shall observe almost as great a change as between the jaws of the chimpanzee and Esquimaux.

Much of our food today is prepared in such a manner that mastication is almost unnecessary and arrest of development of the jaws must follow as a result. While the etiology of all dental anomalies cannot be accounted for by arrested development of the osseous structure of the jaws, many of them originate in this manner. Others are caused by some structural changes in the early development of the teeth and jaws.

We will first consider the impacted lower third molar. This condition is in the large percentage of cases caused by either lack of normal development, or by arrested development of the inferior maxillae. The second molar immediately in front and the coronoid process behind bound the space accorded to the third molar. Each tooth which has no deciduous predecessor is developed beneath the base of the coronoid process, that is, the first, second and third permanent molars. The only manner in which these teeth are able to take their places in the normal arrangement of the teeth in the jaw is by the physiologic absorption of the anterior surface of the coronoid process. While this process of absorption is going on, on the anterior surface, through the laws of compensation, nature has provided for a deposition of bone on the posterior surface. Through these two processes, absorption and deposition of bone, respectively, all of the molar teeth are able to erupt into their proper positions.

However, when either of these physiologic processes in the process of development of the mandible is arrested, the interval between the anterior surface of the coronoid process and the last erupted tooth which is usually the second molar, with be insufficient for the normal arrangement of the presenting third molar. Under these conditions, the tooth in pressing forward, takes a direction in which the least resistance is offered to its progress and a contition becomes manifest which we term impaction.

Another condition which must be recognized in accounting for the malposition caused by the impaction, is that the tooth has been directed from its course at a comparatively early period of development, irrespective of resistance offered at the time of eruption. This condition may be accounted for by a change in the character of the cancellous bone immediately adjacent to the developing tooth. If, through some inflammatory process peculiar to the individual or by the same process set up through some pathologic disturbance, or trauma, a secondary deposit of dense bone is laid down, around the developing tooth, a change in direction of eruption of the tooth may take place or there may be a prevention of its eruption entirely. This retarded condition is also due to the fact that at the points where all of the other teeth are located, soft spongy bone or alveolar process is found and absorption of tissue takes place more readily at these points than at the point of eruption of the third molars which is made up of dense cortical or true bone. Frequently it will erupt until it comes in contact with the second molar and then stop on account of insufficient space in which case only a small part of the tooth will be presented. The upper third molar presents an entirely diffirent condition on account of the difference in the character of the bones in the upper and lower jaws. When, for any reason, there is a retarded development of the upper jaw which makes it shorter in its anterior posterior diameter, so that there is not sufficient room for the normal eruption of the third molar, the crown of that tooth will point toward the cheek, taking the direction of least resistance in its course. It slides out to the side, being guided by the second molar, which is already in place. If there were plenty of room both upper and lower third molars would erupt without pain which is rarely ever the case.

The cuspid teeth are the next in the series most likely to be impacted. The etiology of this condition is the same as that of the impacted molars—viz. insufficient space for the normal presentation of the tooth in the process of eruption. The cuspid teeth make their appearance long after the eruption of the lateral incisor and sometime after the eruption of the first bicuspid. In case of retarded development of the jaws, there may not be sufficient room between the lateral incisor and first bicuspid tooth for normal eruption of the cuspid tooth and a resultant impaction occurs.

On account of the tendency of an erupting tooth to follow the course of least resistance,

the tooth will slide out to the buccal or into the lingual surfaces of the jaws. It is also true that at an early period of development of these teeth, that a secondary deposit of bone in the jaws, due to an inflammatory process, may influence a change of direction in the developing tooth so as to change its position entirely when it erupts.

While the third molar and cuspid teeth are predisposed to impaction more frequently than any others, yet any tooth in either jaw may become impacted if the environments are not such that normal eruption can be made possible.

The next anomaly which we will consider is the unerupted or aberrant tooth. This condition may be present in any part of the jaws and any tooth may be the offending member.

There is always a reason for the tooth being unable to erupt normally. The developing tooth meets some obstruction in its normal course such as a supernumerary tooth or a normal tooth which has previously erupted and already occupies the space belonging to the erupting tooth. Again, a previous inflammatory condition in the cancellous bone may have set up secondary deposits of dense bone which will prove an obstruction to the developing tooth and deflect it out of normal alignment. This subject would not be complete without a few words relative to the supernumerary tooth.

"Any tooth in excess of the normal number of thirty-two, although clearly cases of reversion of type, in many instances, are included in the category of supernumerary teeth." [1]

Albrecht and others maintain that the incisor teeth were originally six in number and thus a supernumerary lateral incisor would be clearly a reversion of type. Their appearance in any situation is evidence that the normal number of tooth buds has been exceeded.

Guilford divides these teeth into two types, those having typical anatomic forms and those having the conical form.

Supernumerary incisors in either jaw having typical forms are not uncommon. In the upper jaw supernumerary centrals and laterals both may appear, the latter more frequently. Supernumerary teeth may occupy any position relative to the dental arch but are more frequently seen on its lingual surface. In addition to molars and incisors supernumerary bicuspids are occasionally found, while supernumerary cuspids are very rare.

1. Burchard.

PATHOLOGIC CONDITIONS PRODUCED.

We will now consider some of the pathologic conditions which are induced by these dental anomalies. We will take up first the impacted lower third molar.

Perhaps the most common disturbance we encounter is local infections of the soft tissues surrounding the tooth. This condition is usually present in the partially erupted tooth when the gum tissue which should occupy the space between the anterior surface of the coronoid process and the third molar has been forced out over the distoocclusal surface of the tooth and becomes contused in mastication. Bacteria laden saliva and food particles are forced into the interstice between the tooth and swollen tissue and infection takes place. Inflammation is set up and maintained, which is not limited to the injured parts, but more commonly extends to the adjacent structures involving the soft textures about the ascending ramus and frequently involving the paratonsillar region.

Deglutition becomes painful and trismus is set up. Many times the patient is unable to open the jaw more than two or three millimeters. After a time suppuration takes place and the movements of the jaws become less constrained.

In these acute conditions, it has been the writer's experience that immediate procedure is contraindicated. In his opinion the treatment of choice is to apply counter irritants, the application of cold compresses or ice to the face, and have all of the body eliminating processes active.

After the inflammation has subsided and the jaws have become less constrained, conditions should be restored to normal by surgical procedure to obviate repeated attacks. This condition will be rarely found around other impacted teeth than the lower third molar.

A pathologic condition which may occur as a result of any impaction is pressure resorption. When the crown of one of these teeth is lodged or impacted against the root of the adjacent tooth, the hard enamel surface of the impacted tooth will cause a resorption of the tissues of lesser resistance of the other one. This may go on to such an extent that the nerve pulp of the adjacent tooth will be encroached upon.

A very serious condition may arise from an impacted lower third molar by pressure being exerted upon the inferior dental nerve. The position of these teeth in the jaws predisposes

to impingement upon the inferior dental canal. In such cases a reflected pain may be set up which will be expressed in any part of the head which has its sensory nerve supply from the fifth or trigiminal nerve.

Neuralgia may have its etiology in such conditions. The late Dr. Henry S. Upson, former professor of neurology in the Western Reserve University, ascribed many of the nervous disorders which "mankind is heir to" to impacted teeth. He states that "certain types of nervous disturbances caused by dental impactions has almost the clearness of a laboratory experiment, as in it the severest symptoms are set up by the simplest irritant. Pain may be from the beginning to the end quite lacking." It is the constant though mild irritation, perhaps not sufficient to produce pain, that sets up some of these nervous disturbances which may of themselves take on a violent form.

Alopecia areata, or baldness occurring in sharply defined patches, leaving the scalp smooth and white, is a condition due to a nervous disturbance. The impacted tooth must be considered an etiologic factor in this affection.

During the last few years, we have had several cases of this nature in the University Hospital that showed marked improvement after the removal of impacted teeth.

Another abnormal condition is quite frequently found associated with the presence of impacted lower third molars is a tendency for these teeth in their effort to erupt to force all of the lower teeth forward. Orthodontists have found it almost impossible to retain normal occlusion following orthodontic treatment with these teeth present in the jaws. Many fine results have been ruined by the presence and activity of impacted teeth.

What are the pathologic possibilities of the unerupted tooth? It is a remarkable fact, and one which has not been fully explained, that unerupted teeth having lain dormant for years in the jaws suddenly become the seat of purulent inflammation with sometimes serious symptoms. Such cases are by no means rare. Some writers are of the opinion that under certain conditions these teeth may act as foreign bodies and may even fall a prey to resorption. Under these conditions where an irritation has been set up and purulent inflammation has become seated, a bone abscess may form around the region of the unerupted tooth. This abscess may develop until a large portion of the jaw becomes involved.

Another condition which is frequently seen in mouths of men and women under thirty years, is the cystic growths connected with teeth whose eruption is retarded. While in the light of our present knowledge the explanation for the formation of these cysts is largely theoretical; Tomes has given the writer the most plausible theory. He states that when the development of the enamel of the tooth is completed, its outer surface becomes perfectly detached from the investing soft tissue and a small quantity of transparent fluid not uncommonly collects in the interval so formed. This fluid ordinarily is discharged when the tooth is erupted, but when from some cause the eruption of the tooth is prevented, it increases in quantity and gradually distends the surrounding tissues, causing a resorption and disintegration of the osseous structures. These cysts may go on developing until a large portion of the jaw is involved.

Again, the unerupted tooth may, by coming in contact with the roots of the normal erupted teeth, cause pressure resorption and thus produce a permanent injury to them. When these teeth lie in close proximity to a nerve trunk, they may cause undue pressure and set up the same obscure nervous disturbances which have been attributed to impacted teeth.

The last of the dental anomalies which we are considering in this paper is the supernumerary teeth. One of the most common resultant consequences of the supernumerary teeth is the obstruction they present to the normal eruption of the permanent teeth. They may retard or entirely prevent the eruption or they may erupt first and force the normal tooth to erupt out of alignment. Warnekros has written quite extensively on these teeth as an etiologic factor in the causation of cleft palate. It is a well recognized fact that in cleft palate there is not a lack of palatine tissue, but a failure of union of the tissue. Warnekros believes that the failure of union is brought about by excessive number of teeth. He says

> "As the normal number of teeth during their development requires a comparatively large space, it is explicable that in very many instances, a supernumerary tooth, with the limited conditions of space in the intermaxillary bone which is not yet completely ossified may itself cause a cleft formation in the intermaxillary bone."

In support of this contention which he says he no longer brings forward as an hypothesis, but declares to be a fact, he cites an extensive series of cases as proofs in which he emphatically states that a supernumerary tooth can al-

ways be proved to have been the cause of the cleft.

While we are not yet ready to accept the deductions of Warnekros in their entirety, there seems to be some relation between these anomalies and cleft palate. We frequently find that supernumerary teeth are present in these cases. The writer, however, is not able to follow the reasoning of Warnekros in clefts of the lip alone or in clefts that do not involve the maxillary processes. It has always been a question with the writer as to whether the supernumerary tooth is the etiologic factor in cleft palate, or whether the changes which take place in embryonic life which result in cleft palate do not in some manner affect the development of the normal number of teeth.

DISCUSSION.

DR. CYRENUS G. DARLING: After listening to this very able paper one wonders what can be done to avert this terrible disaster which is coming to the human race. Every once in a while we learn of a new calamity, and still the race goes on. The question arises whether it is worth while to attempt to develop the human jaw back to the chimpanzee shape with all the teeth and strength, or, if this is advisable, whether it can be done. We evidently are departing from this line of development because I can recall very clearly in my early childhood that there was a silver quarter with a hole in it to put a string through, and this coin was kept in the family to tie around the neck of the children while the family was cutting teeth. Another thing in those days was the method of feeding. When the child began to take food, and that was as soon as he could sit up, he was given a crust of bread, or when the parson called and there were many chicken bones, these were saved and given to the babies to use in his mouth as he saw fit, and these agencies no doubt were very largely responsible for the development of the teeth and jaw. We all know in our work on cleft palate and repair of the jaw how very little pressure it takes to mould the shape of the jaw. There is another thought which comes in here and that is the higher the civilization the more of these cases of lack of development will be found. In this lack of development there is pressure upon the nerves which may cause most any nervous disorder and serious disease. When we advance to a stage of civilization we can say that people who have advanced to such a stage where they spell culture with K are especially developed along these lines, resulting in many things with which we are familiar.

DR. JAMES G. VANZWALUWENBURG: Naturally, I am more or less interested in these conditions because I am often a factor in their discovery. I have almost reached the point where I am not surprised to find a tooth almost anywhere. They are the most ubiquitous things we have about the face.

## "LYMPHATIC DISEASE IN CHILDREN"

HAROLD DEB. BARSS, M. D.
(From the Surgical Clinic, University Hospital, Ann Arbor, Michigan.)

In examining the records of the Surgical Clinic a short time ago, I learned that recently seven children had been operated upon for suspected appendicitis, and at exploration certain unexpected and strikingly similar features were disclosed. A review of the case histories of these seven children brought to light these common findings.

All the symptoms occurred in children under puberty.

All presented a syndrome suggestive of recurring attacks of appendicitis. Thus—In each, abdominal pain was the most prominent feature, coming in attacks lasting from a few hours to three days. Each complained of nausea. Constipation was the rule. Unlike the usual picture of appendicitis, no case had fever or a leucocytosis. The urine and blood tests were in every case normal. The Wassermann examination of the blood was negative in all cases. A gastrointestinal X-ray in five of the cases gave a picture of ileal hypomotility very typical of chronic appendicitis. And in no case could any pathology be discovered for the appendiceal trouble.

Yet at operation in each and every case the appendix was found free, without any adhesions, and somewhat hypertrophied. It did not look very pathologic. The unusual feature was the presence of enormously enlarged lymph glands in the mesentery and along the spine, retroperitoneal. Those glands draining from the appendix seemed to be especially affected. In each case the appendix was removed, and some of the glands. Again there was unanimity in the reports from the pathologist, for in all he wrote, "Lymphoid Hyperplasia in Appendix and Lymph Gland." Twice he suggested the possibility of a lymphatic constitution.

In trying to correlate this syndrome with other known diseases, that of status lymphaticus seemed most nearly to approach these seven cases. This disease is spoken of as, "A rare condition met with chiefly in children in which the lymph glands and lymphoid tissues throughout the body are in a state of hyperplasia." The lymphatic structures of the alimentary tract are most commonly affected. We believe that in some way our cases are instances of a degree of lymphatic constitution which fortun-

ately are not of an aggravated type and have not resulted in the so-called, "thymic death."

We cannot say positively that the appendix is at fault; but we do know that after removal of the appendix, all the symptoms from which the children had been suffering for two or three years were completely relieved. In two cases a second X-ray after operation showed that the intestinal motility had become normal. What effect appendectomy will have on the general glandular hyperplasia we cannot as yet predict. In favorable cases we would expect atrophic changes in the thymus and lymphoid structures to take place as the child grows older, and the patient may then outgrow all evidence of the disease.

This then seems the rational procedure;—appendectomy to relieve the immediate active manifestations of the disease. This to be followed by ultimate disappearance of the disease through natural retrograde changes.

DISCUSSION.

Dr. Cyrenus G. Darling: This is a very interesting study. How or why were these lymphatics enlarged? And why did the pathologist say that the pathologic condition was struma? When the tonsil becomes infected we think nothing of it if the neighboring lymphatic nodes become enlarged; in fact, we rather expect them to be enlarged and we say that if we remove the tonsils that the nodes will probably return to a normal conditi n. In the child it is not impossible to suppose that some of this same type of infection may get by the tonsil and be carried along the intestinal tract, evading the destruction which might possibly come to it in all this journey, and safely reach the appendix, there to enter the lymphoid tissue of the appendix and be distributed to the mesenteric lymphatic system from that point. In some of these cases we find quite a wide distribution in the mesentery, wider perhaps than would come from the direct invasion from the lymphoid tissue in the appendix. From the appendix we are told that the lymphatic nodes are found in the mesoappendix and that we might expect the infection from the lymphoid tissue to be carried to these lymphatics and thence to still further lymphatic distribution in the mesentery of the small intestine. It has been our observation in nearly all cases of appendicitis to find that there are two or three enlarged lymphatic nodes over the iliocecal junction, and in some of these cases we are able to trace this enlargement of the lymphatics toward the median line, that is, over into the mesentery and upward toward the median line. Yet, in some of these cases we find a wider distribution than would seem to follow this line unless there was some blocking of the lymphatic tract, and by the damning back of the lymph channels, other lymphatic nodes became involved. It is perhaps more logical to think that there has been an invasion of Peyer's patches or the solitary glands in the intestine from the same infection that invaded the appendix and its lymphoid tissue, and that this lymphoid tissue had been the point of infection, and that the infection had been carried to the lymphatic nodes. There is one point in this that we don't clearly understand, which may have some bearing upon this point, that is the effect of irritation of the appendix upon the iliocecal valve, whether this does at times produce a spasm of this valve with retention at the lower end of the ilium the same as retained material in the appendix may cause infection of lymphoid tissue there. May it not by spasm of the valve and retention in the lower end of the ilium, cause infection of the lymphoid tissue in this area; or, on the other hand, if there is a patent iliocecal valve which may allow the material to lie still in contact with the lymphoid tissue, may not infection take place in that way? We find that nearly all of these cases occur with constipation, that the material does lie in contact with the lymphoid tissue of the appendix or the lower portion of the ilium long enough to produce irritation and the absorption of some material which causes increase in the size of these glands. If the lymphoid tissue of the appendix should be the point of absorption, we are doing the right thing to remove the appendix and prevent the absorption. If the iliocecal valve is controlled in any way by inflammation or irritation of the appendix, we do the right thing when we remove the appendix. At least, this is a question where we are able to offer some points in favor of this treatment by the results obtained. And I think that we will do well to procede in all of these cases in this way by the removal of the appendix.

There may be marked inflammations in this locality, and I just recall one which goes still further in this thought of inflammation of Peyer's patches. It was a case of a child having hemorrhage following constipation, with tenderness in the region of the appendix. This hemorrhage and tenderness led to the idea of intususception. The hemorrhage was profuse and not deeming it proper to let this child go without an examination, I opened the abdomen. There was no intususception, but about three inches up the ilium there were eccymotic spots along the outer part of the bowel and these extended over on the one side toward the mesentery and the lymphatic nodes were enlarged. I did not proceed any further with this operation. This patient recovered, and after a week or ten days, the temperature went down. Now, you might say I am mixing up a case of typhoid infection, but I don't believe so. It began with constipation and had marked constipation except for the loss of blood. It didn't run the course of typhoid fever. I believe it was some other infection. I think that something might be done by more careful examination of these glands to determine the type of infection, whether it is an intoxication, or an invasion by some form of bacterium. I think that we should carry these studies still further along that line.

Dr. James G. VanZwaluwenburg: All of this makes very interesting speculation, of course, but up to this point we have to confess that most of it is speculation. It only throws into bold relief the dense ignorance under which we labor in regard to the physiology of this portion of the bowel. It is only another phase of the problem of the causes of ileal stasis. I say these are speculations. In

justification of that assertion I want to call your attention to the fact that Dr. Darling at one time aruges that the hyperplasia is primary and the intestinal condition may be secondary, and the next moment he assumes that it may be the other way around.

I have been very much interested naturally, because I have been studying the motility of that end of the bowel rather intensively and have reached no conclusions. I want to suggest still another possibility which Dr. Darling omitted. One school of physiologists states that the motility of the bowel is largely controlled by chemical factors, by hormones. It seems to me altogether logical to suppose that the lymphatic hyperplasia may in some way modify the absorption of the food elements in the lumen of the bowel, or of the specific hormone, and as a result food is retained there until the requisite amount of these substances is supplied. Experimental physiology teaches that when a bowel is full it is much more active than when it is empty, that is, that the contents of the bowel excite it to greater activity. Yet we find almost invariably that in ileal stasis there is a hypomotility, a reduction in the irritability, or the muscular tone is so much higher that there is a tendency for retrograde peristalsis. But these are purely speculations. We most urgently need greater light on this particular segment of the gastrointestinal tract than on any other physiologic subject with which I am familiar.

# TRANSACTIONS

OF THE

# Clinical Society of the University of Michigan

## Stated Meeting, April 3, 1918

**The President, JAMES G. VAN ZWALUWENBURG, M.D., in the Chair**
**Reported by REUBEN PETERSON, M.D., Secretary**

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## REPORT UPON THE CLINICAL SYMPTOMS AND ANATOMICAL FINDINGS IN THREE CASES OF ORGANIC BRAIN DISEASE SHOWING DISTURBANCES OF AN APHASIC AND AGNOSTIC TYPE.

ALBERT M. BARRETT, M.D.
(From the Psychopathic Hospital, Ann Arbor, Michigan.)

At a meeting of this Society in November, 1913, I presented a patient who showed an aphasic disorder of speech. This case was reported in detail in Volume V of the Transactions of the Clinical Society of the University of Michigan. Since that time the patient has died and we have had an opportunity of studying the pathologic condition that was responsible for the interesting disturbances of speech.

The essential facts of the symptomatology and course may be briefly recalled in this connection. The patient was a woman who, previous to an attack of apoplectiform character, at the age of 57, had been regarded as one somewhat above the average in intelligence, and whose health had usually been good. The apoplectiform attack was transitory in its effects and after a few days she seemed as well as ever. She performed her household duties acceptably until the spring of 1913. At that time there was a very notable impairment in her general efficiency and particularly there was a marked disturbance of her memory. She was abnormally irritable and occasionally made strange remarks that suggested delusions. Whatever difficulty may have been present in her speech at this time did not attract as much attention as did the general mental let-down and it was for this that she came into the Hospital. A summary of the large amount of material obtained in the examinations of the disturbances of speech shows the following: On the receptive side of speech, she understood simple requests and commands but failed in whatever was complex or when several sentences were given in sequence. She would correctly pick out familiar objects that had been named to her. She recognized objects from their sounds and could name most objects that were shown to her. She could read the separate letters of the alphabet and could pick out her name, but was totally unable to read even short sentences. Recognition of objects by touch was unimpaired and a number of substances that were tasted were correctly identified. On the emissive side of speech, the disorder was most striking. There was little spontaneous speech. She would answer questions and occasionally volunteer a brief conversation. Sometimes she could read the alphabet correctly but more often she would stop before it was finished and make no effort to go further, seemingly forgetting what she was trying to do. Sometimes numerals would be intermingled among the letters. The same difficultv occurred in counting. Spelling of words of a few letters was often correct but with longer words she usually failed. Often the number of letters given was approximately right and sometimes the wrong letters had resemblance to those that should have been given. e. g. Tree was spelled t-r-o-o. Michigan, M-i-g. There was no paraphasia in spontaneous speech or in repeating from dictation. Perseveration was rather frequent, especially when she was fatigued. She was totally unable to write. She usually began a task as though she might be able to carry it through but her production had no resemblance to the letters desired. She seemed to have no understanding of gestures or, if she did understand them, she

could not transfer their meaning into the proper action.

There was a suggestion of an apraxic difficulty in executing commands. There was a marked delay as if she were puzzled how the action was to be done. When asked to point to her nose with her right hand she looked at her right hand, moved her arm apparently with some purpose and then placed her hand on her knee and made no further effort. Actions more complicated than this were rarely done correctly.

She understood the use of objects and, while there was usually some difficulty in using them, her actions with these were fairly correct. The main difficulties chiefly concerned the elaborative aspects of speech. In a large measure these were explainable by the profound memory disturbance that was present. There was a marked defect of intelligence. This was seen in her dullness, her loss of interest in the care of her person, and a considerable degree of emotional deterioration.

A summary of this speech impairment showed that there was preserved understanding of the simpler forms of language but impairment of anything that was at all complicated. There was a total alexia, or inability to read. Recognition of objects was preserved. There was no paraphasia. Repetition from dictation was impaired. There was some perseveration. Spontaneous speech was reduced in amount and there were gross errors in spelling.

Clinically, this type of speech disorder closely resembled what has been described as transcortical aphasia, a disturbance of speech in which the difficulty lies largely in the field of elaboration. Such a condition as this is more commonly observed in conditions of diffuse brain atrophy, especially where this involves regions concerned with the functions of speech or paths radiating to and from these. In rare instances, the same results might occur from focal lesions closely bordering the stations for the receptive of emissive functions of speech.

There were present during the clinical course of this case definite evidences of a diffuse disorder of the brain cortex. Such were the emotional dullness, the emotional impairment, and the general intellectual deterioration.

The patient was transferred early in 1914 to the State Hospital at Kalamazoo. The examinations at that institution noted the same disorder that had been found in the hospital at Ann Arbor. Deterioration advanced gradually until she became helpless on account of her general mental weakness. Without any definite apoplectic attack she developed an inability to use the right leg in walking. Her deterioration reached a most extreme degree. She finally became unable to stand alone. While in bed the legs were flexed and her head held stiffly. Gordon's and Oppenheim's reflexes were present in both feet. The pupils were irregular and sluggish in their reaction. There was slight internal strabismus of the left eye. Towards the last the muscles of the body would twitch whenever she moved. Death occurred in February of 1916 from broncho-pneumonia. The entire duration of the disease was about three years.

The examination of the brain showed it to be strikingly small. Its weight was 1130 grams. There was a fairly large cyst of an old internal hemorrhagic pachymeningitis covering the convexity of both hemispheres. Part of the contents of the cyst was from a rather recent hemorrhage.

The hardened brain showed the effects of a lateral compression from the cyst. The convolutions in general were very small, and in the parietal and frontal regions were somewhat flattened. The speech areas did not show more marked gross changes than other regions.

Microscopic examination showed extreme atrophy of the convolutions and the presence of numerous miliary plaques such as are characteristic of senile brain atrophy. The nerve cells showed severe degeneration. Many were sclerotic and generally they were overloaded with fatty pigments. Many nerve cells showed degenerations of the neurofibrils. These were thickened and formed whorls and tangles of the type rather frequently found in connection with the formation of plaques.

The age at which the process occurred, namely 57, as well as the prominence of the changes among the neurofibrils, show that this case was one of the premature forms of senile brain atrophy, known as Alzheimer's Disease. The speech disturbance thus finds its explanation in a diffuse rather than a focal lesion, a finding that is usual for most cases of transcortical aphasia.

### A CASE OF TACTILE AGNOSIA.

The case is that of a man, R. H., S. P. H., No. 2238. At the age of two he had a severe fall and fractured both shoulder blades. He was not unconscious at the time. He was backward in mental growth, not talking until the age of six. About this time, he entered school but his progress was slow. At the age of nine, he was placed in the

state school for the feeble-minded but after a brief time was taken home and again placed in the public schools. At the age of twelve, he had only reached the third grade. As it was supposed that his backwardness was due to some head disorder, at the age of twelve, a trephine operation was made in the left parietal region. He appeared brighter after this but did not return to school. At fifteen, he started to work in a factory. He continued to work successfully, receiving good wages, until he was twenty-seven. He then began to complain of a tight feeling in his head. He grew more dull and at times there were periods of unconsciousness. There were also periods of confusion in which he would wander away and there was amnesia for all that happened during that time. A second operation was performed in June of 1916. In this a large button of bone was removed from the upper part of the left parietal region. Three days after the operation, there developed a paralysis of the right hand and face. Mention was made by his friends of his inability to speak correctly at this time. The paralysis of the hand and the speech disturbance passed off after a week but there developed attacks of Jacksonian epilepsy. For about a year, these occurred about once each week. During these attacks, the right side of the body and the right arm and the face were contracted and jerked violently.

There was usually some mental disturbance associated with these attacks. Recently, the attacks have been more severe and in many of these he bit his tongue and was definitely unconscious.

He entered the Psychopathic Hospital in February of 1918, at the age of 30. Here, he was dull and disinterested in his surroundings. He gave a fair account of his past experiences and description of his attacks. He was usually irritable and complained much of various somatic discomforts.

The neurologic examination showed a circular defect in the bone of the right and left parietal regions, marking the place of the two previous operations. There was a marked dysarthria in the spontaneous speech and with test phrases. There was slight atrophy of the muscles of the right lower arm, the circumference of this arm being about one inch less than the left. The left grip measured 34 on the dynamometer, the right 12. He complained of the right hand being cold and the skin of the hand was shiny and the volume of the muscles was less than on the left side. There was no disturbance of sensibility of the left arm or hand. In the right hand, he appreciated light touch, pain and position sensations, but these reactions were far less keen than in the left hand. At times, following mild degrees of unclearness, all qualities of sensation seemed to be absent in this hand but when mentally clear these reactions were present to a considerable degree. At no time was there any ability to name or identify objects placed in the right hand. When a knife was placed in this hand, he would usually remark that he did not feel anything. Similarly, he failed to name or identify keys, a watch, pencil, or pen. Whenever these objects were placed in the left hand, they were correctly recognized and correctly named.

On one day he complained of peculiar feelings of numbness that passed every few minutes along a narrow strip up his back and out over the shoulder up to the defect in his head. At times there were involuntary contractions of the muscles of the right hand and often he spoke of this arm and hand being numb. There was slight spasticity of the right leg. Both knee jerks were increased and Babinski's reflex was present in the right foot.

These various disturbances were present during the entire period of observation. On the 10th he had a severe convulsion. This began without warning. The head was slowly drawn to the right and the right angle of the mouth was drawn downward. The fingers of the right hand were clenched and this arm was slowly flexed and moved across the body. After a brief period of fixation in this attitude, rapid clonic movements spread over the entire body. These were followed by relaxation and a period of stupor lasting about fifteen minutes. Two days later he had five convulsions. On the day following there were sixteen convulsions and on the next day convulsions followed one another at very brief intervals. During these last three days, he was continuously unclear. Death occurred during a convulsion.

A partial autopsy was held shortly after death, the head alone being examined. The scalp was firmly adherent about the edges of the defect in each parietal bone. In the right parietal region, there was a circular defect in the bone, about 3 cm. in diameter. This had been closed in by a dense membrane. The dura mater in the region of both defects was firmly adherent to the bone and beneath the defect on the left side it was densely adherent with the underlying brain substance. The brain tissue was soft and flabby and flattened under its own weight. The veins were slightly filled. There was no arteriosclerosis.

After fixation, a more detailed examination of the brain was made. The brain substance lying beneath the defect in the left parietal bone was torn and firmly adherent to pieces of the dura mater. The limits of the area affected were rather difficult to sharply outline by reason of the softened character of the tissues. It lay within the lower half of the posterior central convolution, involving the extent of about three-quarters of an inch of the convolution. The anterior central convolution was not involved and it could be easily separated away from the dura and then showed no defect.

Of particular interest in this case was the lack of ability of the man to recognize and identify objects that were placed in his right hand in spite of the preservation of the perceptions of touch, pain and muscle sense. Such a disorder has been designated astereognosis. This condition has always been one of much interest from the standpoint of localization and interpretation of the psychic features that were involved in the process. The disorder is essentially an inability to form a correct concept of the object, even though the elementary sense qualities, such as touch, muscle, sense and pain, furnish information. It, therefore, is a disorder of association, a disturbance of the synthesis of elementary sense qualities into a concept. An interesting question is whether the disorder is one of associations between the differ-

ent sensory elements themselves or between these elements as a whole and areas of the cortex in which are stored memories of other sense perceptions, such as visual, auditory or possibly other special sensations, the inter-relation of all of these being essential for the awakening of the idea of the object. Such a disorder would be produced by a lesion cutting off the central convolutions from the occipital or temporal areas of the brain.

On this point, our case furnished no conclusive information. The localization of a lesion to produce disorders of this type always lies back of the central fissure. This lies either in the posterior central convolution in the middle third or in the parietal region where it may cut across association paths between the receptive areas for the sensations of touch, pain and temperature and the region concerned in visual impressions. In the case under discussion, the lesion involved the cortex and closely adjacent white substance of the lower part of the middle third of the left posterior central convolution.

It did not seriously disturb the anterior central convolution. It did not seem deep enough to cut across the deeper association paths with the visual areas and its effects seem rather to be among the areas of primary sense perception. However, it does not definitely prove this.

The close proximity of the lesion of the head area at the lower end of the central convolution may explain the involvement of the head and face, symptoms which were present during the early part of the course, and may also explain the aphasic difficulty that was noticed for a brief period.

### A CASE OF MOTOR APHASIA.

The case is that of a woman of average intelligence, who could read and write. At the age of sixty, she had a sudden attack of faintness. A few days later she had difficulty in talking. She could not recall the word she wished and in her speech many words were used incorrectly. Although she recognized members of her family, she could not speak their names. This difficulty with speech increased, until her speech was limited to replies of "yes" or "no" and a few familiar words. During this time she was somewhat more dull than formerly, but she was able to do her housework acceptably.

She was admitted to the State Psychopathic Hospital in November, 1917. The physical and neurologic examinations showed slight enlargement of the heart to the left. The blood pressure was 140. The gait was unsteady, and she swayed in Romberg's position. There was slight asymmetry of the face. The pupils were irregular in outline but the light reactions were not impaired. In both feet there was a slight dorsal flexion of the great toes. There were no paralysis or sensory disorders.

There was no spontaneous speech. She understood all that was said to her, but her replies were limited to a few words, always spoken with difficulty and in low tones. She gave her age and her name, but to most questions there was no response at all, or sometimes she would repeat the question. When asked if she knew the correct answer, she nodded affirmatively. Efforts to have her read were without success. She would take the copy, look at it and make no response.

While usually mildly stuporous, she would rouse during examinations and show a fair interest in what was being done. This stupor increased rapidly until, one week after admission, she became unable to feed herself and was too feeble to stand. There was no longer any spontaneous speech, but her reactions all showed correct understanding.

Neurologic symptoms became more marked. The right arm became paralyzed. A well marked Babinski reflex was present in the left foot but not on the right. On several occasions she vomited. The Wassermann reaction was negative on the blood. Unfortunately, there was no examination of the fundus.

Her stupor deepened and death occurred nineteen days after admission.

The autopsy showed evidences of increased intracranial pressure and the left hemisphere was more voluminous than the right.

After fixation, section of the brain showed a tumor mass occupying the subcortical white area and much of the overlying cortex of the anterior two-thirds of the Island of Reil. Anteriorly the area reached into the posterior part of the left third frontal convolution. It very definitely infiltrated into the subcortial parts of Broca's area. Its internal border extended along the outer margin of the claustrum. Externally, it infiltrated the pia of the Island, and had a small extension into the tip of the first temporal convolution. The posterior part of the first temporal convolution as well as its connections with the Island were not involved.

The tumor was found to be a glioma. It was of an infiltrating character and in several places there were cysts filled with products of disintegration.

In summarizing the disturbance of speech, it is found that this entirely concerned the emissive functions. These were not entirely abolished but were very severely impaired. Understanding of spoken language was preserved. Her ability to read was abolished. Clinically, it corresponds very closely to the motor types of aphasia.

### DISCUSSION.

DR. MAX PEET: I recently had one case which showed beautiful asteriognosis. This was a boy whom Dr. Darling had about two years ago with a tumor on the left side pretty close to the median line in the post central gyrus. At the first operation this was diagnosed as sarcoma. At the second operation it was diagnosed endothelioma. His asteriognosis did not clear up after the first operation. Another case recently showed one of the other symptoms which Dr. Barrett has mentioned, a case of injury to Broca's area. This was a boy with endothelioma which grew down from the dura, but had not invaded the cortex at any point. This was purely a question of injury to the motor speech center from pressure without. This boy did not have the complete motor speech loss. If you asked him how old he was he would start counting, one, two, three, four, five, six, six, six, eight, ten eleven, and could not go beyond that. If you helped him out and said twelve he would say yes, but could

not go beyond. If you said nineteen, twenty-two, etc., he would shake his head. When the number twenty was mentioned he would say yes, as this was his age. At times he could count farther than at other times. This was before we removed the tumor. His hearing was perfectly good, he understood every word you spoke to him showing that the posterior part of the speech area was intact. And his associations were all right. His only trouble lay in his inability to go beyond a certain line. On the other hand, he could say expressions which he had been in the habit of saying for a long time. He could say, "O dear." Very often he said this when something bothered him. Some of these patients cannot say a word but they can swear beautifully. Then we had another case which illustrated an injury to all three of these areas. The man had paralysis on the opposite side also. He could see perfectly well. There was a question whether he could hear but he could not say a word or understand a word, and yet could understand any motions. He had some optic atrophy and we had difficulty in telling whether he had visual aphasia. We submitted to him a newspaper with enormous headlines, "Kaiser asks for peace." I knew any individual who would get the visual meaning of that would show some interest. He showed none. These particular cases of Dr. Barrett it seems to me, are especially interesting because they bring out so clearly the possibilities we have in brain localizations, and the aphasias and asteriognosis are very important in cerebral surgery, giving us very definite information as to the location of many tumors. Fortunately many of my cases have been those in which the tumors have been on the surface, although most of the cases are gliomas below the surface. Decompressions would prevent loss of sight and relieve pressure, but would not relieve the symptoms, but rather make them worse if the function was not entirely lost.

I am rather at a loss to say why any simple operation such as the one described by Dr. Barrett should leave asteriognosis or any other marked lesion in the brain cortex. If the dura is opened and brought together again we do not expect trouble from adhesions. On the other hand, when we do a decompression and remove the bone the adhesions between the brain cortex and overlying muscle very seldom give paralysis or loss of sensation below it.

Dr. Theophil Klingmann: I would like to call attention to the fact that these cases are of unusual interest because they are clear-cut, both clinically and anatomically, and for that reason are of great help in correlating the symptom complex with the anatomic changes. These cases are very satisfactory and for that reason are very encouraging in this line of work. The attempt to correlate mental symptoms with anatomic lesions has met with some success and appears more hopeful as far as solving many of the clinical manifestations on an anatomic basis.

Dr. L. H. Newburgh: I would like to ask Dr. Barrett if there is usually some other lesion in this precocious senile dementia i. e. if there is any renal or cardiac disease and general atrophy? I ask this because every now and then there are cases of precocious arteriosclerosis described that primarily involve the heart and kidneys. I was wondering whether there is any connection between such cases and the case described here. Might it be the same process?

Dr. Barrett: As a rule, the arteriosclerosis process occurs at an earlier period than the process of senile brain atrophy. As a rule, arteriosclerosis of the brain is associated with focal lesions such as well defined softenings or atrophies. In this case, we have a process entirely different. It is not certain as yet what the factors are in producing these peculiar plaques. In the greater part of these cases, there is a marked fatty degeneration of the cell and very commonly an increase of the glia groundwork structure of the cortex. It is possible that the plaque has its beginnings in the accumulations of products from cell disintegration in the meshes of the glia fiber. Very often, there is at the same time an arteriosclerotic disease of the brain but the process of plaque formation and that of the arteriosclerosis are quite distinct. There was little evidence of arteriosclerosis in this case. The blood pressure was 140 and at the autopsy there were no marked findings of arteriosclerosis.

Dr. Van Zwaluwenberg: Is there an hereditary tendency in senile dementia?

Dr. Barrett: We have very little definite information on this point. Undoubtedly, there is a tendency in some families for brain atrophy to occur more frequently than in others. There seems to be a considerable amount of evidence that arteriosclerosis runs in families. One very commonly finds among patients suffering from arteriosclerotic brain disease a history of cardiac and vascular disorders among the ancestors.

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## REPORT OF A CASE OF TRANSVERSE MYELITIS FROM A BULLET WOUND.

Charles L. Washburne, M.D.
(From the Surgical Clinic, University Hospital, Ann Arbor, Michigan.)

Case No. 10278, male, age 21, German-American. Entered the Hospital, February 28, 1918.

*Chief Complaint.*—Paralysis of left leg.

*Family History.*—Father living at fifty-five and well. Mother living and well, age forty-eight. Four brothers and six sisters living and well. No history of tuberculosis, heart disease, nephritis, cancer or insanity in the family. No hemophilia.

*Personal History.*—Measles, chickenpox, mumps. No other diseases. Neisser and lues denied. Smokes and chews in moderation. No history of previous injuries.

*Marital History.*—Was married at eighteen. One daughter living and well. No history of miscarriages. Wife died of pneumonia one year ago.

*Present Illness.*—Began December 4th, 1916. The patient's brother was showing him a new gun which he had purchased, at that time having been recently appointed to the police force. The gun was supposedly empty at the time, and the brother was about to snap it shut when it discharged, the bullet entering the abdomen at a point some four or five finger breadths above the umbilicus and three finger breadths to the left of the median line. The

bullet did not come out posteriorly. The patient immediately became stiff and was laid upon the floor to await the arrival of the police patrol which took him to the hospital. The accident took place at 6:30 in the evening. An exploratory operation was done two hours later. The liver was found to be grazed but there was no puncture of the intestines. The bullet was not removed, the patient being told that it would give him no further trouble as it was probably lodged between the eleventh and twelfth ribs near the spinal column. The patient was paralyzed from the waist down at the time of the accident but, after five weeks in the hospital, he regained complete control of the right leg. The left leg has remained almost completely paralyzed, with slight improvement. There is still sense perception in the left leg and sensation is more acute than during the first few weeks following injury. The patient remained in the hospital foi nine weeks. A dull aching pain, which the patient says began at the point of entrance of the bullet, has been slowly going down the leg and is now located at the ankle. The patient's left foot feels cold to the touch. The ankle is slightly less movable than the right. During the early period of his injury, the patient had no control of the sphincters. At the present time he has poor control of the bowel and is unable to hold urine longer than two hours. He was catheterized twice daily while in the hospital. He sleeps well during the day but is kept awake much during the night by pain in the left leg.

*Physical Examination.*—Pupils are central, equal and regular. The left reacts to light more readily than the right. No nystagmus, extraocular movements normal. No enlargement of the cervical glands. Respiratory movement free and equal, liver dullness about normal, no rales. Heart rate, 90. Percussion normal. There is a large scar some eight inches in length extending from a point four or five finger breadths above the umbilicus and two or three finger breadths to the left of the median line to a point on the level with the umbilicus. The incision was made through the bullet wound which is in the center of this scar. Abdomen otherwise negative. Knee jerks not obtainable. The legs are thin and show muscle atrophy of disuse. The left leg shows no muscle action.

*X-ray Examination.*—Reveals a bullet in neighborhood of the second lumbar vertebra. Stereo plates of this region demonstrate this foreign body opposite the second lumbar disc, apparently in the neural canal, anterior to the cord. The outlines of the centra both above and below are smooth. The posterior arches are intact. There is no evidence of injury to the bony structures to indicate the tract of the projectile. Evidently this bullet has entered from the side or through the fibro cartilage. A Wassermann taken of the blood, was negative. Blood pressure, systolic 110 m. m. hemoglobin 100 per cent. reds, 5,000,000; whites, 8,600.

March 4, 1918, operated under ether anesthesia in the Orthopedic Service. An incision was made extending from the twelfth dorsal to the third lumbar spinous processes. The muscles were carefully separated from the spinous processes and laminae. Hemorrhage was checked by packing the wound with adrenalin gauze. The interspinous ligament was cut at the third lumbar, the spines cut with a bone forcep and turned upward. The laminae were resected and the cord coverings exposed for a distance of about two inches. The meninges between the first and second lumbar bodies were thickened. A hard substance could be felt on a level with the second lumbar vertebra deeply embedded in the posterior wall of the cord covering. Further dissection showed this hard substance to be a very much distorted lead bullet. A portion of the bullet was intimately associated with the nerve fibers of the cord from which it was removed with some difficulty. A free flow of spinal fluid followed the removal and the cord was seen to pulsate normally. No area of cord degeneration could be demonstrated. The dura was closed with No. 00 chromic catgut. The muscles and interspinous ligament were sutured with No. 2 chromic catgut, continuous sutures. Skin sutures were of silk worm gut. A small rubber tissue drain was inserted for serum drainage. Sterile dressings were applied and patient returned to bed in good condition.

*Post Operative Record.*—Patient reacted well from the anesthetic. Two hours temperature; 100, pulse 128, of good quality, respiration 28. For first 24 hours the foot of bed was kept elevated. For five days following operation the patient complained continually of a severe headache which was only slightly relieved by ice caps and large doses of aspirin. The morning following operation, patient had not voided and complained of pain over bladder; thirty ounces of urine were removed by catheter. The drain was removed at the end of 48 hours and the wound showed no leakage of fluid. On the sixth day the patient began to void urine normally without any dribbling. During the first week there were several involuntary bowel movements but none thereafter. There was a daily p. m. temperature of 100 to 102 for first six days. The temperature reached normal on the seventh day and remained so. Stitches were removed on the twelfth day at which time the wound was healing satisfactorily. During the third week there was severe pain in the left leg which disappeared in a few days. At present he has perfect control of the sphincters. He has been up in a wheel chair for five days and on crutches for the past three days.

*Physical Examination*: April 2, 1918. Left upper thigh shows three inches atrophy. No atrophy of calf. Tapping on left patellar tendon causes pain, no reflex contraction obtained. Right patellar reflex shows very slight action. No ankle clonus or Babinski on right. No ankle clonus on left. Plantar irritation gives no response whatever. Left foot and leg covered with a profuse perspiration. Patient is unable to move left lower limb. No muscle action above knee. Slight action in muscles controlling foot. The left foot assumes a position of equinus deformity. There is anesthesia to pin point along the internal surface of the left foot. No other area of cutaneous anesthesia, although sensation is much diminished over the whole left lower extremity. Incision of back well healed

showing only a few remaining crusts. Pressure sore over sacrum and left buttock nearly healed.

DISCUSSION.

DR. MAX PEET: I think Dr. Washburne is to be congratulated upon getting this bullet out without doing any more damage to the cord than had been done by the original trauma. Bullets are especially difficult to remove on account of the deformity which catches onto the adhesions, the various nerve roots and the cord itself. Personally I believe all such cases should be operated upon as soon as possible, but often a year afterwards perhaps some improvement, or complete cure can be hoped for. There are in many cases a pure paralysis due to pressure. In such cases removal of pressure will give relief. One of the great advantages of early operation comes from the relief of the tension in the cord. Experimental work has shown that, where a definite trauma would always produce a lesion from which an animal would completely recover, a slightly greater trauma would produce a lesion which would always give permanent paralysis. But when the cord was incised in the latter cases the animal would recover complete motor and sensory function. So in these early cases, the earlier the operation the better. If the cord is swollen, bluish, and non-pulsating, simple incision into the cord will often give relief. The fact that this man has not as yet recovered use of the one leg is no sign that he is not going to. Six months is the average time for improvement, and some cases show improvement for two years. I think Dr. Washburne can look for still more improvement in this case.

DR. JAMES G. VANZWALUWENBURG: I showed the lantern slide reproduction of this man's radiograph at a previous meeting of this society, and I commented that I could not conceive a bullet of such size getting into the spinal canal without producing damage to the bony wall. The only way I could conceive is that it must have dodged in from the side, being deflected in the abdomen and passing in between the laminae or pedicles, and if it did, it is curious that it did not injure some of the spinal roots.

DR. WASHBURNE: This man had his bullet for fifteen months before he came here. He had entirely healed up and didn't know that he had a bullet. Now he thinks it is there. What are your indications? What would you want if you knew you had a bullet in such a place and were paralyzed? Would you want to leave it there? It might do no good, to operate, but you would never be satisfied until it was out. As a matter of fact, this bullet did not lie against the cord, but below the lower end of the cord. It evidently came in from the side, following the lamina. One portion lay right on the fibers from the cord and was in contact with the anterior wall of the spinal canal. The root fibers of the cord lay right on top of it. When the bullet was removed the spinal fluid came out after it. The main part of the bullet lay anterior to the axis of the canal.

# ETIOLOGY AND TREATMENT OF DYSTOCIA OF CERVICAL ORIGIN WITH A REPORT OF FOUR CASES.

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Dystocia as a result of a rigid cervix is a well known although relatively infrequent, obstetric condition. To a mild degree, however, it is present in nearly all primiparous labors, but here it is physiologic and does not ordinarily require treatment. This paper is confined then to those cases in which there is a marked delay in the dilatation of the cervix.

The distinction of functional and organic rigidity is convenient but open to criticism because there probably is no true functional rigidity. In all of the cases of cervical dystocia reported here there is an organic element.

Inasmuch as the internal os is usually partly dilated before the onset of labor, the difficulty is met with the external os. Here, according to Galabin & Blacker (1), there are strong circular muscle fibers which may contract tightly and give rise to serious trouble. This is especially likely to occur in nervous women in cases of reflex irritation where there are frequent examinations or premature attempts at delivery. Fieux, quoted by DeLee (2), denies the existence of circular muscle fibers in the cervix uteri at term and ascribes all these cases to anatomic causes. While this may be so, nevertheless there are numerous cases recorded in the literature in which the cervix remained tightly contracted despite hard pains, in the absence of any known etiology.

In premature rupture of the membrane the elastic dilating wedge of the bag of waters is lost and we may get spasm resulting in poor dilatation. The cervix may become swollen and edematous and in extreme cases may even slough off. In old primiparae the rigidity is probably anatomic rather than spastic. Here the elastic tissue has been more or less replaced by fibrous tissue which dilates with difficulty.

The treatment of functional rigidity of the cervix is bound up with the treatment of organic rigidity because it is difficult and even impossible in some cases to determine which factor is more important. Whenever there is difficulty in the first stage a careful vaginal examination is indicated in order to determine the cause of dystocia. In the spastic type of rigid cervix pressure of the finger tip against the os, especially during a pain, may serve to relax the spasm and allow dilatation to pro-

ceed normally. If not, the finger may be inserted inside the os to separate any adhesions which may exist between the membranes and cervix. If this is unavailing we may administer chloral by mouth 15-30 grains which may be repeated if necessary. Morphine ⅙-¼ of a grain by hypodermic often is efficacious in relaxing the spasm. Whether these drugs act by relaxing the spasm or not, there is abundant clinical evidence to mark them as valuable drugs in the management of a tedious first stage. Atropine alone in doses of 1/100 grain or combined with morphine (3) has been recommended in this condition. Hot vaginal douches, Sitz baths, the application of cocaine to the cervix and the injection of glycerine (4) into the os have also been recommended. Our present tendency, however, it is to avoid soiling the vagina unless operative interference is indicated. If these measures are not sufficient to secure dilatation there is undoubtedly an organic basis for the dystocia and we will proceed as will be presently described under the treatment of that condition.

Organic rigidity of the cervix may be due to a variety of causes. Of the congenital malformations, complete atresia is incompatible with conception and hence needs no discussion here. Longitudinal septa in the canal from failure of complete fusion of the Mullerian ducts are not very common but may seriously obstruct labor. Congenital hypertrophy of the vaginal portion of the cervix is rather rare and usually these patients do not become pregnant. Its effect upon labor is the same as the acquired form of hypertrophy which is more common. This usually comes on during pregnancy when it grows quite rapidly. T. C. Smith (5) reports such a case in which the long thickened cervix did not dilate well. He finally applied forceps and was able to extract the child using his instruments as dilators.

Inflammatory processes about the cervix may act in two ways in bringing about dystocia. In the first place we may have a chronic endocervicitis with a fibroid deposit or we may have adhesions formed between the membranes and the lower segment of the uterus. In this condition the external os may be practically obliterated by an inspisated plug of mucus. This produces a condition known as conglutio orificii externii. The adhesions counteract the dilating tendency of the uterine contractions and we often get premature rupture of the membranes. This gives us an added factor in producing delay. The cervix becomes thinned but does not dilate. We can almost always locate it by speculum as a small depressed area. Simple pressure against the external os may overcome the resistance after which dilatation goes on rapidly.

Malposition of the cervix may interfere with the normal progress of the first stage. Uusually the os is deep in the posterior fornix with the anterior fornix obliterated although occasionally the reverse is the case. The etiology of anterior displacement of the cervix is usually sacculation of the anterior uterine wall due to pregnancy in a retroverted uterus. Sacculation of the posterior wall of the uterus in a case of anteversion will displace the cervix backward. This is especially prone to occur when pregnancy follows such operations as anterior fixation or interposition operations for prolapse. The treatment of this condition depends on the individual case. Occasionally expectancy is rewarded by spontaneous dilatation. If not the finger may be hooked in the external os and an attempt made to pull it forward or backward, as the case may be, into its proper position.

Syphilis has been described by Tarnier as producing rigidity of the cervix in a variety of forms, namely, the induration of the primary sore, hypertrophic mucous patches such as occur during pregnancy, gummata, tertiary ulcers and finally a diffuse sclerosis comparable to syphilitic stricture of the rectum. The majority of writers on this subject have tacitly accepted syphilis as a cause of cervical rigidity. However chancre of the cervix and secondary lesions are the only authentic syphilides which have been found in this locality and they leave no residium. Tarnier and others who have reported these cases made their observations before the discovery of the Wassermann test and the spirochete and consequently their conclusions cannot be given serious consideration. Dr. Wile, in a careful review of the subject, finds no authentic case of tertiary syphilis of the cervix. Syphilis of the internal genitalia of the female is practically unrecognized and its role as an etiologic factor in dystocia cannot be estimated until we know exactly what lesions it produces. Undoubtedly it affects the pelvic organs including the cervix but the older methods of diagnosis have failed to reveal it so it remains for the demonstration of the spirochete to establish the true status of syphilis in obstetrics and gynecology.

New growths of the cervix, notably polypi and carcinoma, may obstruct labor. Both of

these conditions however are usually associated with sterility. Their effect upon labor and the treatment is not within the scope of this paper and will not be considered here.

We have finally cicatricial stenosis of the cervix and it is chiefly with this type of obstruction that I wish to deal. We may have scars in the cervix as the result of old lacerations, following cauterization or as a sequence to operations such as trachelorrhaphy or amputation.

The healing of old lacerations may result in the deposit of scar tissue which at a subsequent labor will not dilate under the influence of uterine contraction. Such a case is reported by Lusk (6). The patient was first seen after having been in labor for a considerable length of time. She had had one child before but there had been no operations. Examination showed that the cervix was undilated and the uterus tetanic with a well marked contraction ring. Before she could be prepared for Cesarian the uterus ruptured and the woman died.

Strong cauterizing medicines and even the actual cautery were used many years ago for such gynecologic conditions as erosions, eversions, and ulcerations of the cervix and consequently dystocia occasionally resulted from the scar tissue. But the cervical cautery is no longer used except in the management of cancer and as a result this cause of prolongation of the first stage is no longer seen.

The effect of trachelorrhaphy upon subsequent labor is a subject which has been considerably dealt with. The consensus of opinion is that the operation properly performed will have little obstructive effect upon the character of the next labor, There are, however, several instances reported in the literature in which there was considerable dystocia and one of the cases reported here comes under this classification. Probably in these cases, as has been suggested by Mitchell (8), there has been too much cervical mucosa removed in the repair resulting in a small os. Theoretically, simple repair of the lacerated cervix should not produce dystocia. The scar which results is linear and if this has been properly covered with mucous membrane the amount of undilatable fibrous tissue will be reduced to a minimum. Leonard (9) reports ten cases of pregnancy following trachelorrhaphy only two of which had difficult labors. He quotes fifty-one additional cases collected from the literature in only six of which was dystocia encountered and rightly concluded that the operation properly performed has little influence on the cause of subsequent childbirth.

Amputation of the cervix as a cause of dystocia has been fairly extensively studied by the French but the American literature shows only one valuable contribution on this subject. Leonard reviewed the literature in 1913 and added eleven cases from the Johns Hopkins clinic. In seven of these labor was very difficult and he concludes that the operation should be avoided during the child bearing age if possible. Doleris(10), in reporting seventy-eight cases operated by himself, claims that with perfect technic the scar tissue will be reduced to a minimum and dystocia will not result. Although this may be true to a certain extent we can hardly call the technic of the Hopkins' clinic to account for the large percentage of cases of dystocia following their method of amputation.

The difficulty in dilating such an os is readily understood. The scar from an amputation is circular surrounding the os and, when it contracts as all scars do as they grow older, its tendency is to still further diminish the caliber of the canal. Normally the force of the contractions of the uterus are exerted at the point of least resistance which is the internal os. At this point only is there an absence of external pressure. This action is further enhanced by the tendency of the uterus to draw up into the fundus the fibers of the cervix. Added to this we have the hydrostatic action of the bag of waters. But the smaller the diameter of the opening the less purchase the uterus and the advancing part have upon it and if this is further reinforced by a circular band of scar tissue, severe dystocia is almost inevitable.

Amputation of the cervix has another influence which has been especially emphasized by Audebert (11) & Leonard. There is a marked tendency to abortion and premature labor. Of thirty-two pregnancies reported by the latter, 17 or 55 per cent. terminated prematurely, consequently in considering the management of these cases we must bear this possibility in mind. Both amputation and trachelorrhaphy apparently predispose the patient to sterility. This, as has been pointed out by Johnson (12), is more apparent than real because so many of the patients when operated upon are reaching the age when pregnancy is unlikely. Leonard has shown that the tendency to sterility is more common after amputation than after trachelorrhaphy, because stenosis is

more likely to occur when the resulting scar is circular.

The course of labor in cicatrical stenosis depends upon the amount of scar tissue present. In some cases the external os will remain small for a considerable length of time but finally the scar tissue gives way and the cervix dilates rapidly. Occasionally, as in a case reported by McPherson (13), the cervix may tear at the side and if operative interference is delayed delivery may occur through such a tear. If the os is very resistant it may present at the vulva still undilated. Rupture of the uterus and death from shock, hemorrhage or peritonitis will occur if the stenosis cannot be overcome. In some cases the cervix may become markedly edematous due to interference with the blood supply and after a time it may slough off. Hemorrhage from this complication may be serious and even fatal. Sepsis is a real danger partly because of the bruised edematous tissue and also because interference is usually necessary in these cases.

The chief danger to the child is asphyxia. This is especially marked when the membranes rupture prematurely because, in that event, the hydrostatic cushion of the bag of water has been lost. If operative interference is instituted, we have the added danger of forceps injuries. Provided there are no complications present, the prognosis is uniformly good if the patient is seen early in labor and the proper measures are taken to safeguard both mother and child. If the first stage is unduly prolonged or if ill advised operative procedures are attempted, the outlook is not good.

In the treatment of cicatrical stenosis of the cervix we should bear in mind that although intelligent expectancy may be followed by spontaneous dilatation, yet many babies have been lost and mothers exhausted by fruitless attempts to overcome the obstruction. It is true that there is little danger to the child during the first stage of labor if the membranes are unruptured but in the prolonged first stage of dystocia of cervical origin we must keep careful track of the condition of the child and be ready to interfere at any time in its interest.

Should interference be necessary we have our choice of manual or instrumental dilatation, multiple incision, vaginal hysterotomy and abdominal Cesarian section, either the classical operation or the Porro modification. The procedure will depend upon the condition of the case. Manual dilatation is often very difficult, due to the scar tissue in the cervix. Instrumental dilatation is a questionable procedure in any case because of the difficulty in gaging the amount of force used. Multiple incisions of the cervix are of great value in those cases where the internal os is completely dilated and the external os is undilated. In such a case incisions from 1½ to 2 cm. in length will cut only the supravaginal portion of the cervix and not endanger the bladder or the rectum. The anterior and posterior incisions will be practically bloodless but the lateral ones will give rise to hemorrhage in some cases. This can be controlled by pressure of the advancing part until the child is born after which sutures are applied, or the incisions may be made between clamps which are removed after several minutes. If this is done there is rarely very much hemorrhage. Mason (14) speaks highly of this method and reports several successful cases.

Vaginal hysterotomy may be used if the child is not too large. It is especially valuable where the cervix is not completely taken up. However, it is very hard to do when the cervix is far back and cannot be pulled down. Cesarian section has been used when conditions contraindicate vaginal delivery. If the classical operation is done it is essential to see that the cervix is open sufficiently to ensure proper drainage of the lochia. LePage (15) has reported a case in which the Porro Cesarian was performed in order to obviate this difficulty. It may also be done in order to prevent the possibility of future pregnancies, or in case the uterus has been infected by attempts at delivery from below.

The following cases have been selected from the Gynecologic and Obstetric records in order to illustrate some of the above points.

Case 1. Mrs. G., a Greek housewife of 26, (Obstetrics No. 1328) was examined at the fifth month of her first pregnancy. Her history and examination were entirely negative. The cervix was long and the external os small. When eight and a half months advanced her membranes ruptured prematurely and pains began three hours later. Examination showed the fetus to be in occiput left anterior and the child in good condition. Under the influence of moderate pains the cervix dilated to the size of a quarter in about six hours. After this she began to have a constant bloody discharge but examination revealed no other sign of placenta previa or premature separation. After twelve hours of hard pains the cervix was found on rectal examination to be thick and swollen and about one-half dilated. The patient was anesthetized and mid forceps applied. The cervix was pushed back over the advancing head and the child extracted. It was in pallid asphyxia and was resuscitated with some

difficulty. The anterior lip of the cervix sloughed off subsequently but the mother otherwise made an uneventful recovery. The etiology of this case of dystocia probably was premature rupture of the membranes resulting in cervical spasm. There may also have been an endocervitis with adhesions between the cervix and membranes. The long continued pressure of the head upon the undilated os resulted in interference with the circulation and edema.

Case 2. Mrs. B., (Obstetrics No. 1465) entered the Hospital in labor, October 13, 1916. She had four children living and well and there had been numerous miscarriages. Seven years previously she was operated on for laceration of the cervix. Five years ago she had a miscarriage early in pregnancy. At the time of entrance she had been having very hard pains for ten hours and because the doctor could find no evidence of cervical dilatation he brought her to the Hospital. Vaginal examination revealed the cervix to be obliterated and very thin. The external os was about the size of a dime. After this the cervix dilated rapidly and within two hours the child was born without interference. The rigid cervix in this case was due to a previous trachelorrhaphy. Its rapid dilatation was the result of the giving way of the old scar tissue because examination at the tenth day revealed a bilateral laceration which was quite deep on the left side.

Case 3. Mrs. E. W. Housewife. Married. Was operated upon in June, 1911, for cystocele and rectocele. The Watkins interposition operation was performed, the cervix being repaired and the perineum built up. She was not sterilized. Three years later she entered the Hospital pregnant, about at term. She had been having severe hemorrhages for three weeks. Examination revealed the presence of a complete placenta previa with the cervix pushed far back in the hollow of the sacrum. It was so far back that even with a normally implanted placenta the uterine contraction would have been unable to dilate it. Realizing the impossibility of a vaginal delivery an abdominal Cesarian was done and the uterus removed with the product of conception. This case also teaches us that whenever the interposition operation is done in the child bearing age the patient should be sterilized.

Case 4. Mrs. L., aged 35 (Obstetrics No. 1700), entered the Hospital during the sixth month of her pregnancy. She has four children living and well, the youngest being ten years of age. She was operated upon in 1912 for lacerations of the cervix, at which time an amputation was done. She has always had menstrual difficulty but following her operation this has been prolonged. Severe cramp-like pains begin before the period and last until the flow has ceased. The present pregnancy began in June, 1917. In the latter part of October she began to notice a protrusion from the vulva, which increased in size until it was as large as an orange. This interfered with walking and became markedly irritated. She could not urinate until she replaced the mass. Examination showed the presence of a six months pregnancy complicated by prolapse. The bladder came down outside of the vulva and the vagina was completely inverted so that the fornices were only a centimeter inside of the vulva. The cervix could not be found at first but careful inspection revealed a tiny os which just admitted the finest probe. It was identified by a small amount of stringy mucus which was present. It had been amputated and the stump covered by vaginal mucous membrane thus accounting for the difficulty in locating it. The prolapse could easily be replaced but recurred as soon as she stood on her feet. It could not be held in place by a pessary because of the extensive perineal laceration. She went into labor a week before the expected date of confinement, her pains beginning at six p. m. of March 17th. At first they were ten to fifteen minutes apart but by midnight they were coming at four minute intervals, lasting about forty seconds. She was put upon her feet and the cervix pushed outside of the vulva. It was found that there had been no attempt at dilatation. When the prolapse was replaced, if she remained in bed, there was no tendency toward recurrence even during a pain. She continued in labor throughout the night, the pains becoming harder but no more frequent. At ten a. m. she was examined by Dr. Peterson who found that there had been no change in the cervix. Inasmuch as sixteen hours of reasonably hard pains had produced no effect upon the cervix, it was decided to interfere. The patient was anesthetized, and a probe inserted into the cervical canal. It was just large enough to admit a uterine sound. This was followed by a small hemostat and dilated until the metal dilator could be introduced. This was replaced by the thumb and forefinger as soon as possible. Dilatation was continued normally until the thumb and three fingers could be introduced. At this time it was found that the internal os also was incompletely dilated. Manual dilatation was unusually difficult owing to the scar tissue so the bladder was dissected up off the uterus and the anterior lip was split in the midline for a distance of about ten centimeters. The child was then delivered by version and extraction. It was a female weighing six pounds two ounces and was only slightly asphyxiated. The placenta was then expressed and the incision repaired. The patient has made an uneventful recovery, her highest temperature being 101.6 degrees on the third day.

The choice of interference in this case is worthy of explanation. In the first place, the question of the abdominal route presented itself. This was rejected for the following reasons: 1. The cervix and vagina had been prolapsed outside of the vulva for six months and undoubtedly were badly contaminated by pathogenic organisms. 2. The external os could be found only with the greatest difficulty by direct inspection. Had an abdominal Cesarian been done it would have been necessary in order to secure drainage to dilate the cervix, which would have been even more difficult to find from above. This difficulty could have been obviated by the Porro Cesarian but this would interfere with subsequent operative procedure to cure the prolapse. 3. The adhesions following Cesarian, the possibility of which could

not be removed, would seriously interfere with a subsequent attempt to cure the prolapse by the interposition operation. For these reasons, then, the vaginal route was chosen.

Manual dilatation was first attempted but was rejected after trial because of the scar tissue about the os. Multiple incisions were rejected because the internal os was likewise not completely dilated and according to Dührsen their use is not applicable in such a case because of the proximity of the bladder and rectum. Consequently vaginal hysterotomy was performed. There is only one disadvantage which can be found with the operation in this case. There will undoubtedly be scar tissue along the attachment of the bladder to the uterus. Whether this will interfere with the interposition operation remains for the future to decide. This patient will return for the correcting of her prolapse sometime after the process of involution is complete.

There is one other point which deserves mention in connection with the last case and that is the occurrence of procedentia complicating pregnancy. In these cases there is a great tendency to abortion but if pregnancy continues the prolapse usually retracts itself spontanneously. The development of the condition during pregnancy, however, such as occurred in this case, is very rare and the fact is noteworthy that she spent most of her time after the fourth month with a mass the size of a large orange outside of the vulva without interfering with the pregnancy.

The treatment of cystocele during labor is also brought up in this condition. If the patient lies down there will be no interference with the bladder during the first stage. It should be emptied frequently and we must caution her against bearing down. As the head descends after dilatation of the cervix the cystocele may be tucked back out of the way.

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DISCUSSION.

DR. CYRENUS G. DARLING: There are one or two points in this paper which might be discussed, and one is the question in regard to the formation of cicatricial tissue following cervical repair or amputation. I suspect that the statistics that have been gathered in this paper belong to a past age in gynecology, as no one doing operations of this type nowadays, would expect his operation to be followed by marked cicatricial tissue, hence these reports as to the cause of dystocia would hardly stand with present methods of operating. The methods which may be pursued to overcome these various contractions are properly mentioned and most of them can be followed by most any one who practices obstetrics even in a general way. Occasionally there will be a case such as the last one described which will require a more difficult and careful operation. Fortunately these conditions are rarely found, at least, I take it to be so. I never encountered one in five or six hundred confinements.

There is one point which might be raised, and that is in regard to amputation or repair of the cervix causing sterility, or rather, causing miscarriage. I think that is hardly true if the operation is carefully performed; in fact, it is very frequently performed to overcome this very condition, and if sterility occurs it would be because of the imperfect operation, or perhaps infection following such.

DR. HENDERSON: Trachelorrhaphy especially is an operation done for the cure of sterility. Occasionally a very marked bilateral laceration of the cervix may be the cause of repeated abortion. The repair will probably result in the carrying of future pregnancies to full term. Amputation of the cervix, however, would never be done for this purpose. The operation of amputation of the cervix is not always properly done and many of these cases become infected and in these cases there is always excess of scar tissue. These cases will occasionally be met with by the obstetrician.

# TRANSACTIONS

OF THE

# Clinical Society of the University of Michigan

## Stated Meeting, May 1, 1918

**The President, JAMES G. VAN ZWALUWENBURG, M.D., in the Chair**
**Reported by REUBEN PETERSON, M.D., Secretary**

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## A NOTE CONCERNING THE EPIDEMIOLOGY AND TREATMENT OF AMEBIC DYSENTERY WITH A REPORT OF TWO CASES.

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Amebic dysentery has in the past been reported and discussed in this Clinic because of the apparent rarity of the condition in these latitudes. At present such is no longer the conception, but conditions abroad, which will sooner or later be brought home to us, warrant a review and make a discussion distinctly worth while just at this time.

The troops from the tropics are carrying their flora of animal parasites with them to the lines in France, and the spread of the infections is especially favored by the intimate relations and exigencies of camp and trench life. England has already reported the inevitable effects; great numbers of British soldiers have become infected, consequently incapacitated and sent home for convalescence. At this point the problem has begun to assume a position of unusual importance, for the ordinary limits of the geographical distribution of the tropical diseases are being widely extended and not only the military, but the civilian population as well, is being threatened. Amebic dysentery is undoubtedly the most important, the most dangerous, and the most resistant to cure of the tropical parasitic conditions to which our soldiers will be exposed. It is of especial interest to us because of its, by no means infrequent, troublesome presence in our midst in the north temperate zone even under the ordinary conditions, in times of peace.

The recent occurrence of two cases of amebic dysentery in this Clinic, brings up two questions of vital importance along the lines of public health for the future. The questions are, first, is the disease transmitted by carriers; and second, are there sporadic cases arising in the north temperate zones?

Extensive investigations by eminent parasitologists such as Craig (1) of the U. S. Army. Fantham (2) of the British Army and others have recently added many new and important ideas to our knowledge of the life history of the entameba histolytica and the mode of spread of the parasitic infection. The vitally important question of carriers of amebic dysentery was worked out conclusively by Walker (3), who found that by feeding the encysted forms of entameba histolytica to twenty men, he was successful in infecting eighteen, four of whom developed the typical dysentery, and the other fourteen became the so-called "contact carriers" of the organism. The amebae appeared in the stools on the average about six days after the ingestion. The term, "contact carriers," has thus been applied to those carriers who have never been affected with the disease, while the term "convalescent carriers," is applied to those who have recovered from the disease but continue to expell the cysts.

Dobell (4), Dale (5), Fantham (6), Imrie (7), Inman (8), Waddell (9), and others definitely demonstrated that carriers were responsible for the continuance of diarrhea among many of the soldiers for a long time after they had returned to England. The cysts were found in the stools of the convalescents as well as in the dysentery patients. Thus it stands a settled question with substantiating proof that there are "carriers" of amebic dysentery.

Low (10) reported very completely a case of amebic abscess of the liver in a patient who had

had no symptoms since his original attack twenty years before. Libman's cases also give long quiescent periods in their histories between the original attack and the liver abscess formation. Such evidence substantiates the supposition of long latent or quiescent periods in the diseases.

In collecting cases from the north temperate zone, we find Sanford (11) of the Mayo Clinic heading the list with 819 cases, 284 of which were due to entameba coli. These, together with Dock's (12) case and those of Walsh, Libman, Tuttle (13), Patterson (14), Rosenberger (15), total about 1,038 cases, 512 of which were due to entameba coli.

Thus 500 cases with three from our own Clinic, (12), (16), (17). (18), (19), of patients residing north of 40° latitude (Philadelphia), who had never been any further south and who had never consciously been exposed, are known to have had entameba histolytica dysentery. This is proof enough that amebic dysentery is not confined to the tropics but that sporadic cases are by no means uncommon in the north. In fact Axtell reported a case of amebic dysentery contracted in the arctics of Alaska. He had a patient who had been a mate on a government boat on the Tanana and Yukon river. In this case the man is supposed to have contracted amebic dysentery by drinking swamp or surface water in the spring of the year. The patient asserted, according to Axtell, that he knew of thirteen or fourteen others with the same trouble. Careful examination with the proctoscope showed amebic ulcerations in which entameba coli were found.

To reiterate and recapitulate, we know that there are carriers of amebic dysentery, that the infection can remain latent for long periods of time and that the disease arises indigenous in the north temperate zone. May it not be possible that the entameba histolytica is an obligative parasite rather than facultative or a saprophyte? That is, the carrier, the living animal body, is the factor in the epidemiology, and that the frequent repollution of the source, whatever it may be, is necessary for the spread of the disease. Along with this is the question of how long the infection can remain in a community after the departure of the animal carrier source.

Our present cases might fall in any one of these groups. The facts in the histories are such that they can not be clearly classified, assigned or fixed epidemiologically. But one of the classes must cover them. It makes but little difference under what head we place them. No matter what the grouping, they are of importance to us now and may be more so in the future. If carriers with a latent infection, they are of especial interest, and if they are sporadic, indigenous cases, they are of just as great importance.

The present cases are the first that have been in the Clinic in two years and they entered within fourteen days of one another. They are both typical entameba histolytica dysentery cases and present many points of similarity. Both have been in Michigan for a considerable time, as I will definitely state presently. Both had been in the tropics at some time in their lives. Each had his present trouble or exacerbation begin in 1914, that is, four years ago, when in each case overwork, worry and nervousness precipitated the attacks, which have continued somewhat intermittently ever since. Both showed entamebae histolytica and trichomonas intestinalis in their stools of blood and mucus; and the blood pictures showed an eosinophilia.

The one had been in the U. S. Army for three years in the Black Mountain Expedition through New Mexico, Arizona and Utah in July and August, 1908, and then in Honolulu, Hawaii, ending in 1910, when he returned to Chicago and then to Michigan, where he has been ever since 1910. He had no intestinal disturbance while in the southern states or in Honolulu, nor did he have any until 1914 when his diarrhea suddenly came on. The question is, did he act as his own "carrier" retaining the latent cyst in his bowel for four years, or did he acquire his infection in Michigan; the former seems more rational.

The other, a Greek, had had the original attack of dysentery in Palestine in 1900. He recuperated after five months, went back to Greece and then came to this country. In 1908 he moved to Detroit where he has been for the last ten years. He had no intestinal disturbance after his original attack in 1900 until 1914 when his bloody diarrhea began. Here again the question is, did he act as his own "carrier" with the encysted organism in his bowel for fourteen years, or did he acquire his reinfection in Michigan. Here again the former seems the more rational, more so than in the first case.

The evidence is by no means conclusive, but there is quite a considerable pointing toward the long latent carrier idea in these cases. Two cases are not sufficient grounds from which to draw any conclusion. We can merely say

that the cases agree with the most recent ideas concerning the disease.

At any rate, whether carrier, latent contact or convalescent, or whether sporadic indigenous cases they should serve to put us on our guard that we may early solve the problem and prevent the maturation of a serious epidemic.

CASE 1. The first patient, Mr. P. B., age 29, a brakeman, married, American by birth, with his home at Port Huron, Michigan, since 1910, that is, for the past eight years, entered the University Hospital, May 3, 1918, complaining of a bloody diarrhea with very frequent stools, weakness and general run down condition. He had had closely intermittent and recurrent attacks of diarrhea for about three and one-half years, that is, since the summer of 1914. His family and personal history are negative. He had been in the U. S. Army and had been in the Black Mountain expedition through New Mexico, Arizona and Utah in July and August, 1908. During this expedition the water was very poor, being green "alkali" surface water with a bitter taste. He was stationed in Honolulu, Hawaii, until 1910, that is, eight years previous to his entrance. There was no dysentery during the southern expedition or in the island just at that time and there were no cases in the post hospitals. He was never in the hospital a day.

His present illness began some three and one-half years ago, late in the summer of 1914, when his bowels suddenly became "loose." At this time he was doing hard work and hurriedly eating very heavy meals. There was a certain nervous tension about the "braking" on the local freight train. This combination precipitated the attack. He had no nausea or vomiting after eating. He had about ten or twelve stools per day and each stool contained considerable blood and mucus. He had to quit work and go to bed for about ten days. Salt water injection did him very little good. He did not improve to any great degree, but went back to work anyhow. He would have fair days and bad days. One year later, in 1915, his rectum was examined and he was told that he had ulcers in the lower bowel. These ulcers were examined by the physicain for amebae but none was found. Nevertheless he was given thirty hypodermic injections of emetine and was somewhat improved. The ulcers in his rectum were pronounced healed. Then about two years ago, that is, in 1916, his condition recurred, the diarrhea slowly becoming worse. He dieted without relief. He has worked right along, taking off three to four days each month in which to recuperate. At times his condition has been very severe. He has passed as high as eight stools per hour. He has always been a thin individual, nevertheless he has lost fifteen pounds in the last three and one-half years. His stools always contained blood and mucus while he was having a diarrhea. His appetite has been capricious.

The physical examination was negative except for tenderness over the splenic flexure and the descendens. Proctoscopic examination of his rectum showed no ulcers, but a redundancy of the mucous membrane (prolapsus recti) was noticed. X-ray examination showed only a hypermotility of the large bowel. The barium enema showed a descendens that was distinctly wider than normal and also considerable gas in the small bowel. The stools were watery, unformed, yellowish in color, and with some undigested food particles. There was macroscopically considerable blood and mucus. On microscopic examination swarms of trichomonas intestinalis and entamebae histolytica were found. On one occasion they were very actively motile but subsequently they were found quite sluggish. The organisms were found very motile on one occasion; they were large and on closer observation the ectoplasm and endoplasm could be differentiated, the nucleus was indistinct and many inclosed red blood cells were seen as inclusion bodies. The cells when quiescent were spherical, the ectosarc and endosarc were not so well defined, but the nucleus was more easily seen as a small refractive body with a thick capsule. The patient had septic tonsils and a pharyngitis. The urine showed a few hyalin casts but no albumin. The blood examination showed a secondary anemia. There were 4,100,000 reds, 13,800 whites and 65 per cent. hemoglobin. A differential count showed 20 per cent. small lymphocytes; 5 per cent. large lymphocytes; 62 per cent. neutrophile polymorphonuclears and 10 per cent. eosinophiles. The blood Wassermann was negative; the blood pressure was normal.

The patient was placed in bed on a liquid diet and given calomel and salts. Then emetine hydrochloride, grs. ½, was given hypodermically, one half hour before each meal. At these same times on an empty stomach, 20 grs. of ipecac in salol coated pills was administered by mouth. Thus the patient was receiving 60 grs. of ipecac per day along with 1½ grs. of emetine. On the second day after treatment was begun, the stools were free from amebae and the dose of ipecac was reduced by 5 grs. on each subsequent day. The stools were reduced to three per day and the patient felt much improved and gained three pounds in three days on a liquid diet. He was then put on a soft diet. No blood or mucus was found in the stools after the fourth day and they became quite formed in character.

CASE 2. The second patient, Mr. E. N., age 42, a Ford factory machinist, married, a Greek by birth with his home in Detroit for the past ten years, entered the University Hospital May 17, 1918, complaining of bloody diarrhea, frequent stools and loss of strength. He has had the trouble for four years, that is, since 1914, but it is worse at times. His family history is negative except that his first wife died five years ago of pulmonary tuberculosis. His personal history is negative except for incidents bearing on his present trouble. He went to Palestine about eighteen years ago, in 1900, a heavy, strong individual. Here he drank some water from a pool while he was working along a railroad. His trouble began insidiously with a gradually increasing diarrhea. His trouble lasted for about five months; he lost a great deal of weight and strength. A physician gave him some pills over an extended period and he was able to gain strength enough to return to Greece where he convalesced fairly rapidly. He never had less than five or six stools per day and frequently as many as fifteen during the attack. This gradually cleared up. He came

to America fourteen years ago, that is, in 1904, and has lived in Detroit for the past ten years, that is, since 1908.

His present exacerbation really began a little over four years ago, 1914. His wife had just died and left him with three children. He tried various housekeepers but none was of any account. He worked day and night to provide for his children and worried much over them. Then he married an Italian, who mistreated the step children. This worried the patient. Under the nervous strain his old trouble came on and he had from six to fifteen or more bloody mucous stools per day. He has never had any nausea or vomiting. He complains of a burning sensation in the rectum and anus on passing the stools. This has gradually become worse. His condition is always aggravated by taking alcoholic beverages, such as beer, wine and whiskey. He has lost about twenty-five pounds in weight in the last four years. He visited various physicians but was unable to get anything more than a temporary relief from their medication. No one else in the neighborhood is bothered with this same condition, so far as the patient knows. On entrance the patient was having six to twelve bloody mucous stools per day.

The physical examination was negative except for marked tenderness in the rectum. Proctoscopic examination showed many ulcerated areas about two inches from the anus. There was much mucus and blood in these multiple, small, round, roughly excavated spots. They were very sensitive to the touch, hyperemic and bled easily. There was some edema of the surrounding rectal wall. X-ray examination with a barium enema showed nothing except that the sigmoid and rectum were very narrow. The stools were liquid, light brown in color and contained undigested fat and meat fibers. Microscopically there was considerable blood and mucus. On microscopic examination great numbers of trichomonas intestinalis and a few entamebae histolytica were found. These latter were occasionally found to be very active, corresponding exactly with those described under case one (1).

Urine showed a few hyalin and granular casts but no albumin.

The examination of the blood showed 4,200,000 red cells. 10,700 whites and 85 per cent. hemoglobin. The differential count of 100 cells showed 26 per cent. small lymphocytes, 3 per cent. large lymphocytes, 3 per cent. large mononuclears, 55 per cent. neutrophile polymorphonuclears and 13 per cent. eosinophile polymorphonuclears. The blood Wassermann was negative; the blood pressure was normal.

The patient was placed in bed and a liquid diet given. Calomel and salts were used to cleanse the bowel. The emetine hydrochloride grs. ½, was given hypodermically, ½ hour before each meal. At bedtime 60 grs. of ipecac were given. This was repeated the next night. The stools were reduced to two or three per day on the third day with a liquid diet. The stools on the third day were free from blood and amebae. The pain in the rectum was markedly diminished.

*Treatment.*—Here again, we can not offer unassailable, tried, controlled and proven, original therapeutic measures, but we can review the most recent approved methods and state our own course with the results. Rest in bed is agreed upon as important in the treatment of this condition. The quinine solution injections from 1 to 5,000 up to 1 to 500 are not used to-day as they were some years ago. (12).

An ingenious method of giving ipecac, in large doses, was devised and used in this Clinic some years ago. To prevent the nausea, caused by this drug, a duodenal tube was inserted and ipecac powder in doses as high as 120 grs. could be given through it directly into the intestines without any ill effects. (19).

Emetine hydrochloride then came into vogue and was given subcutaneously in ½ gr. doses, three times a day, until about 18 grs. had been given. Recent work by English workers has shown us that we must not pin too much faith in the efficacy of emetine hydrochloride. Dobell, (21) at the Walton Hospital, has shown conclusively in a large series of cases that, of the cases treated with emetine hydrochloride, 70 per cent. relapse; and of these, all of which are refractive to further emetine hydrochloride treatment, practically everyone responded to emetine bismuth iodide by mouth. Dale, (8) Low (10) and others reported exactly the same efficiency for the drug.

The emetine bismuth iodide is given by Dobell (21) by mouth on a full stomach in 1 gr. cachets, three times a day until 36 grs. are given.

Dale (5) gives a course of 36 grs. by giving one 3 gr. salol coated pill of emetine bismuth iodide every night for twelve consecutive nights.

Low (10) warns, in a foot note, that it is essential that the drug be given as a powder in a gelatin capsule and not in a compressed form, as he has found the "stearates and keratin coated tabloids" passed in the feces, unchanged.

With all this evidence from reliable English sources where abundant numbers of cases have presented themselves, and where a great deal of excellent work has been done along this line, we are very much inclined to give these methods first place in our therapeutic list. Unfortunately we did not have the opportunity of giving these new measures a thorough trial.

We have had some success with our own combination régime. The patients were put to bed. The bowels were cleaned with calomel and salts and emetine hydrochloride, grs. ½, was given three times a day subcutaneously. At the same time, 60 grs. of ipecac powder in salol

coated pills were given daily as a single dose before sleeping time in one case, and in divided doses of 20 grs. before meals in the other case. The ipecac was gradually reduced by 5 grs. per day and the emetine hydrochloride was stopped after ten days. Since both of our cases were chronic with probably partly healed ulcers and pockets in the colon mucosa and wall, which would lodge cysts of the entameba and protect them from action of a drug in the circulation alone, we resorted to the combination. This method of administration was quite satisfactory; the results were apparently excellent.

We feel that the main virtue of the modern treatment lies in the attack of the organism in the bowel itself where it is lodged. The active principle of the drug when brought to the parts by the circulation is not able to reach encysted forms which are buried in scar tissue or sloughing pockets in the colonic mucosa. We have accomplished both ends by the combination; the oral administration would strike from the lumen, while the subcutaneous injections made sure that beginning metastatic foci outside the alimentary canal would not escape medication.

The use of emetine bismuth iodide is said to make the subcutaneous injections unnecessary.

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# A SIMPLE AND UNUSUAL OPERATION FOR PROLAPSE OF THE RECTUM.


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This case is presented for your consideration to-night because in this one instance at least it suggests a possible remedy for a condition which has been rather troublesome to surgeons, prolapse of the rectum. If you consult surgical works for the treatment of this condition you will be struck with the multiplicity of procedures suggested. The very multiplicity indicates the search of the profession for a satisfactory method. For slight prolapse, the cautery has been used; or a modified hemorrhoidectomy has been performed. A cuff of mucous membrane has been removed, as in the Whitehead operation for large internal hemorrhoids. Other men have advocated perineal incisions with plaiting of the rectal wall. The coccyx has been removed and the rectum drawn up and sutured to the sacrum. Intra-abdominal operations of rectal suspension or fixation have been devised. The method perhaps most successful here has been that in which from the perineum, a complete portion of the prolapsed gut is resected and the upper free edge then fastened to the anal margin.

These measures are all rather severe and many are very technical. In certain cases the patient has suffered so great a loss of blood that a major operation is almost contraindicated or if attempted is attended with considerable risk.

The method which I shall portray to-night is not original, but is one which I have never seen in print. It is possible that it has been tried by many others and discarded as impracticable. I learned of it accidentally a couple of years ago in conversation with a surgeon friend of mine. The statement was made that another surgeon of his acquaintance had performed it in a number of cases with unqualified success. The method is so surprisingly simple that one is apt to discount its value for this very reason. And yet because it is simple I hold that it is worth trying. If it works, we have accomplished a desired result with the minimum of risk and discomfort to the patient, and labor for the surgeon. If it fails, no harm has been done, and we have at all events given it the benefit of a trial.

CASE. W. W., age 54, registered at the University Hospital on November 27, 1917. His complaint was a severe diarrhea with blood and pus in the stools. The past history is not significant. The present trouble began in April, 1917, when he noticed a bearing down sensation in the rectum and soon there occurred a slight eversion of the mucosa. In the early summer a diarrhea developed which grew steadily worse until he began to have an evacuation every hour or two during the day and two or three times at night. There was bleeding and a mucopurulent discharge with the stool. Proctoscopic examination showed a very relaxed sphincter with hyperemia and ulceration of a very redundant mucosa. No prolapse developed at this examination. The patient was examined in the Medical Clinic for several days but no cause could be found for the diarrhea other than the hemorrhoids. He was then transferred for operation of the rectal condition. The hemoglobin was low. The patient had been losing weight. The blood pressure was 115. It seemed unwise to attempt any very radical operation on this man and yet something had to be done to stop this severe diarrhea and hemorrhage. On December 17, 1917, the patient was operated upon in the Surgical Clinic. When under the anesthetic, the prolapse appeared and was found to be about three inches in length. The patient's condition did not warrant a protracted operation and it was decided to perform the following ridiculously simple one. The prolapse was replaced and then small punctate incisions were made about half an inch from the anal margin—one anteriorly, one posteriorly, and one on each side. Through these small cuts scissors were introduced and blunt dissection was conducted beneath the skin so as to connect each puncture, and thus make a complete subcutaneous ring around the anus. Through the anterior puncture silver wire (gauge 19) was threaded on each side, the two ends appearing through the posterior cut. The wire was then drawn up and twisted so as to contract the anal opening to a size which would just admit freely the index finger. The ends were then twisted, the excess wire was cut and the ends were tucked beneath the skin. The punctures were so small that sutures were not needed. The wire was completely buried. A sterile pad was the only dressing applied.

Mr. W. was kept in bed on liquid diet for about ten days. For two days after the operation he had no bowel movement; then three and again two. Following this there were several days without any defecation. After ten days he was given a diet with solid food and allowed to be about in a wheel chair. Then he began to have regular movements, averaging two to four a day, sometimes more and at times less. Chalk was administered in an attempt to decrease the diarrhea and it seemed to help. The patient was discharged five weeks after the operation. When discharged he was averaging one and two stools a day. There was no blood and less pus. The patient felt better and began to gain in weight. The prolapse did not re-appear.

Four months after his operation I wrote to the patient to learn of his condition. He wrote that he was improved in health as a result of the operation; the rectum had not protruded, and that he only had a diarrhea occasionally. There is a mucous discharge so that he still requires a pad. This is not surprising when we know how refractory colitis is.

This, then, is the operation and this the result in the one case in which I have seen it tried. I realize that one can not draw conclusions from a single case. I am not yet ready to say that it is surely of value, but I do believe that certain features are of worth. If it should prove in this and other cases to secure a lasting cure, it is eminently worth while, for it is so simple and without risk. It can be performed under local anesthetic if desired; and the whole operation will take but ten minutes.

Should it prove to be but temporary in the relief it affords, I still feel that it is worth a trial, for it is undoubtedly of value in securing a temporary reduction of the prolapse. Then the physician can treat with better hopes of success the ulcerative colitis and the attendant bloody dysentery. Then when the health and strength of the patient has been improved to the point where he can undertake a radical operation with a minimum of risk, it can be conducted with the maximum assurance of success.

This procedure is new, it is simple, it is untried. I submit it for your consideration as of possible value. I would like to learn the experience of others so that it may be adopted as of value or if necessary, thrown into the discard.

# THE PRODUCTION OF CHRONIC NEPHRITIS THROUGH FEEDING HIGH PROTEIN DIETS.

## A PRELIMINARY NOTE.

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The investigations which I am conducting aim to discover the usual cause of chronic nephritis. The kidney's biggest task is presumably the elimination of nitrogenous waste. Might not an excessive effort in this direction sufficiently prolonged end in a scar?

Rabbits fed an exclusive diet of coagulated egg white show albuminuria within three days and casts after a week.

Rabbits may be maintained on soy beans as the sole food for many months. This furnishes a diet containing 40 per cent. protein. Such animals show albuminuria and urinary casts after four or five weeks. The blood urea averages 100 mg. per 100 cc. blood. The kidneys of rabbits that have lived on soy beans for five or six months, present clear evidence of a progressive subacute or chronic nephritis. In addition to the epithelial injury and congestion, there is a diffuse, extensive increase in connective tissue.

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# GENERAL INDEX

TO THE

# CLINICAL TRANSACTIONS

OF THE

## UNIVERSITY OF MICHIGAN

## VOLUMES ONE TO NINE

1909-1918

EDITED BY
REUBEN PETERSON, M.D
ANN ARBOR, MICHIGAN

# PREFACE.

During the war, the Clinical Staff of the University Hospital was greatly reduced in numbers. The remaining clinicians were overwhelmed with routine work, since the number of hospital patients increased rather than diminished. As a result, it was found utterly impossible to continue the meetings of the Clinical Society, and it was voted that a recess be taken until the times were more normal.

This series of Transactions has been concluded with a general index for the nine volumes, in order to make readily accessible the reported work on any given subject or to locate easily any particular case. It is hoped that the index will be found useful.

In making this general index the editor of the Transactions for the past nine years has been surprised at the extent of the medical and surgical field covered. Many rare and valuable cases have been recorded and have thus become a part, if only a small part, of medical literature.

The underlying purpose of the Clinical Society has been to encourage the younger members of the Staff to report their cases and to gain facility in the discussion of medical questions. Without the stimulus of the Clinical Society and its Transactions many rare cases would have waited for that future time to be reported,—a time that usually never comes.

This series of Transactions should be but the forerunner of a better series made possible by the new University Hospital which nine years ago seemed only a dream but is now an assured reality.

REUBEN PETERSON, M.D.,
Editor.

Ann Arbor, December 1918.

# GENERAL INDEX

TO THE

# CLINICAL TRANSACTIONS OF THE UNIVERSITY OF MICHIGAN

## VOLUMES ONE TO NINE

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